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THE CHARACTERISTICS OF A PSYCHOGENIC HISTORY.

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A few years ago the psychogenic theory of certain diseases was undoubtedly popular. The war seemed to have demonstrated clearly enough that experiences which were associated with emotional states could produce symptoms of illness, and no one towards the end of the struggle had much doubt that the most successful methods of treatment for these conditions were those which were openly psychotherapeutic. Concealed psychotherapy such as is provided by massage or electricity was not nearly so successful in its results. The methods of choice were those of vigorous persuasion, abreaction under hypnotism, and psychological analysis and persuasion. As no new phenomena, which had not already been seen in peace time neurotics, were observed among these patients, it was inferred that the future treatment of the psychoneuroses of peace lay in psychotherapy.

For the majority of organic neurologists this view does, I believe, still hold good; but with a considerable number of psychiatrists, with many general physicians and specialist surgeons, and with perhaps the majority of the profession there has been a growing tendency to regard the so-called psychoneuroses as manifestations of physical disorder, as a something which is poisoning or otherwise injuring the higher centres or as a disturbance of endocrine balance, or again as something causing a hurtful reflex such as astigmatism. Now if we were sure of anything in the war cases we were sure of this, viz., that if the patient was allowed to believe that his illness had a physical basis he did not get stably well till he was rid of this belief. If then we were right in our ideas at that time, and if we are right now in thinking that the peace neuroses are identical with those of war, it must be a misfortune that there should be this swing towards organic theory. The history of the neuroses is one which shows continuous alteration between psychological and organistic views. It will be evident later that this very alternation is proof that the mental view is the correct one, but in the meantime the object in view will be to discover whether there are criteria which shall enable us to distinguish between physicogenic and psychogenic disease.

There is little doubt that some loose thinking in the matter has arisen from the careless use of certain words—functional and organic, neurotic and psychotic, mental and physical. Functional and organic are commonly used as opposed to each other; functional, neurotic and psychogenic are sometimes considered interchangeable; neurotic is considered a smaller edition of psychotic—*e.g.*, the expression, 'the minor psychoses'—and so on. It is submitted that many of these uses are productive of confusion of thought. There is no intention of giving definitions here, a procedure that would merely provoke controversy, but a few observations may not be out of place.

'Functional' and 'organic' are not opposed words at all. Every organic disease produces disturbance of function, and it is this disturbance of function of which the patient complains; thus in heart disease the patient may not complain of his heart at all but of some disturbance of function elsewhere, such as dyspnœa. On the other hand in a purely psychogenic disease, such as hysterical paralysis, there may be physical changes such as cyanosis of the limb.

On many grounds it is not likely that neurosis is a minor edition of psychosis —and why "minor"? A neurosis can be as wrecking to happiness and efficiency as a psychosis. I should say that the difficulties of the neurotic are the exaggerated difficulties of all normal people; the difficulties of the psychotic seem to me to have little affinity with our ordinary conscious difficulties. A speaker not long ago said that he could cite two people, one of whom was in terror at the thought of crossing an open field, the other was in constant though unjustifiable fear that he would be poisoned by some one, and he—the speaker—could see no reason why one should be called neurotic, and the other psychotic. At first blush there is none; but in practice the difference between the two is enormous. The first can be reasoned with on the matter; he will agree that the thing is ridiculous. Such a consideration may not cure him, but it can be discussed with him in the terms we all know and use. The second cannot be reasoned with at all if he is a psychotic: his ideas on the subject are on a plane where none of us can follow. If he can be reasoned with on ordinary lines he is not psychotic on this point.

With the word neurosis there is a further difficulty. A disorder for which no gross lesion can be found is apt to be called a neurosis. Such a disorder may obviously be either psychogenic or physicogenic. Here the word is used to mean a psychogenic disorder, in which there may be physical changes or not; if there are, they are secondary; the word will here be interchangeable with psychoneurosis.

The ideas underlying the words 'mental' and 'physical' still seem to cause endless difficulties, but only because certain physicians desire to be metaphysicians. We are told again and again that because certain organic diseases, such as myxœdema and general paralysis, cause mental symptoms, and because all mental operations must be accompanied by physical change, therefore all symptoms must be indicative of physical changes could we only

have means fine enough to detect them. This subject will be dealt with frequently throughout the paper. Here it will suffice to say that the difficulty is a philosophical one. It is however, convenient for practical purposes to treat mind *as if* it existed, to treat mental operations *as if* they really occurred. Everyone does it in daily life without difficulty. It is hoped that this paper will justify this procedure ; we may do so without resorting to any philosophical speculation as to whether the world is really monistic or dualistic.

No one denies that mental causes have bodily effects ; everyone agrees that grief causes tears even though great names can be quoted in support of the view that it is the tears which cause the grief ; everyone believes that an unpleasant sight or idea may induce a faint ; it is even common ground that such things may be the starting point in time of a neurosis. The opponents of psychogeny, while granting these things, would say that from the standpoint of practice they are unimportant, that the morbid states would not have continued unless there were some overwhelming physical agent at work, and that the cure of the patient depends on our ability to eliminate the adverse influence of these physical agents. We may concede forthwith that psychic traumata do not account for the continuation of symptoms ; they are only precipitating events. But this does not imply the necessity of the presence of the physical if the symptoms are to continue. For proof of the organic view, cases are cited of long-standing nervous disease cured by physical means where psychotherapy had failed ; and those who uphold it do not weary in demonstrating patients where previous observers have been led astray by a ' psychogenic history.' To the first argument the answer is easy, for while psychotherapy sometimes fails it also sometimes succeeds, and therefore its existence is justified : and further it is certain that the administration of any remedy is accompanied by the suggestion that it will bring about cure. It is, therefore scientifically impossible to eliminate the chance that psychotherapy has been used. But the second argument is worth a more detailed answer. I have often heard reports of cases where the doctor had been led astray by this kind of history, but very little discussion about what constitutes a history of the kind we are postulating. It has usually meant that there was some antecedent shock, worry, or grief, the sort of thing that occurs to everyone. If this were to be the criterion there are no cases of illness of any kind which could not be regarded as psychogenic, for everyone has had shocks ; but we must not forget there is no person living, well or ill, in whom an industrious physiologico-anatomist could not equally find some abnormality. If certain people are finding psychogeny where it does not exist, others are finding physical disease where it is of no importance. The truth about every patient no matter what he is suffering from is that we should study him from both aspects, from the physical and from the mental. We shall find something amiss in both, and then we must exercise judgment, decide what is important and what should be neglected. There are many patients whose mental state will be best aided by physical treatment, e.g., a sufferer from duodenal ulcer who is afraid of what

may happen to him. There are others, such as those suffering from hysterical paraplegia, who are best treated by psychotherapy alone, and for whom the extraction of over twenty teeth—as I have recorded elsewhere—was of no benefit, but decidedly the reverse. Some there may be who will require mental and physical treatments, but there will be very many who will require one or the other, but not both. For the purpose of differential diagnosis thorough and careful clinical examination will be approved by all. But it will not be enough to keep us from error. The presence of a psychogenic history will not assure us that the patient is suffering from a neurosis only, but its absence should make us certain that we are not dealing with such a condition, provided we have taken the trouble to ascertain that it really is absent. It may be laid down as an absolute rule: *Do not diagnose a neurosis in an obscure case unless a psychogenic history is forthcoming.* There are some obvious instances, such as hysterical paralysis, where such a precaution is unnecessary, but the rule should be absolute where there is doubt. Not only will serious error be eliminated by such a rule, but the doctor who observes it will get away from the common conception of neurosis as a negative thing, as a collection of symptoms which are causeless and need never have existed, a view which is most deleterious, and leads to no interest in and diminished chances of cure for these sufferers.

Case 1. Mrs. N., age 32. A year before coming under observation she became suddenly sleepless and remained without sleep for a week; since then she had never felt well. Her sleep became better but never certain; she became apprehensive and restless. There was some difficulty in eating, which was worse in the presence of other people. Later she became depressed. For some months she presented no physical signs of disease, and on that account it was judged that she was suffering from a psychoneurosis. It was alleged that she had became tired of the management of her house and that this illness was an attempt to escape from it. Her own feeling was that she could not do it, but she thought that the accusation might be just, and she accordingly became more and more depressed.

Why up to one particular day she had been quite happy in the discharge of these duties, and why on the night following that day she had suddenly become so disgusted with them that she could not sleep, was not demonstrated; but of course her dislike might have been of long standing if only it were unconscious. This sort of guessing is apt to be called psychoanalysis, a specialty that has enough sins of its own to carry without this kind of thing being added to its burden.

Now if painstaking physical examination is to be *the* method by which the diagnosis between functional or organic is to be decided, patients like this one will often be labelled neurotic; but indeed there was no justification for the label if the observer had looked further afield. Up to the time of the illness this woman had lived an ordinary healthy life; there were no stories of obscure illnesses; any difficulties she had had she had met adequately. There was no history of any special anxieties or worries at all. The illness had come 'out of the blue.' The most fanatical psychogenist could have

had nothing at all to offer in the way of psychogeny. On the history the case was organic because of this absence of psychogeny; and so in a few months it proved to be, when the signs of Parkinsonism stamped its encephalitic origin. The patient had been looked at only in transverse section. She had nervous symptoms and no physical signs. Therefore it was held that she was neurotic. We must learn to look at patients in longitudinal section. They have all biographies and we must learn to be biographers. The word biography is used advisedly. It is not a mere history of previous illness or of the family health that is wanted. The biography begins with an account of the family and how they all got on together, what church they went to, and whether they had children's parties, and so on through life. To get an outline does not take as long as it sounds, certainly not longer than the repeated physical examinations that are made in an obscure case.

When the habit of doing this has been acquired it will be found that the psychoneuroses are not so much symptoms of disease as symptoms of a kind of personality. Further it will emerge that though many of the patients had exceptionally unfortunate experiences in life, in others nothing out of the usual had happened; that sometimes they met their difficulties badly but sometimes well, but with a growing inability to overcome them. On the whole the healthy person tends to find his difficulties lessen as he grows older, for he has acquired skill in dealing with them; the neurotic seems to find them more and more difficult, as if what he had acquired was loss of confidence. The normal is in increasing degree influenced by his victories, the neurotic by his defeats. The healthy person from time to time has symptoms, lassitude, lack of confidence, headaches, nights of indifferent sleep, but they do not last long; in the other the symptoms tend to last longer till they get so continuous that the syndrome gets itself called an illness. There is from this point of view no qualitative difference between the normal and the neurotic: the difference lies solely in the mass and duration of the symptoms. The healthy-minded on the whole tends to get over his difficulties by himself, the neurotic to rely more and more on something, on parents, on friends, on dogma, on systems of health, on a doctor. None of these agents need have acted rationally when they succeeded in helping; it did not matter much what the doctor's treatment was so long as he was felt to be a support; when he became tired of the case, or in some other way ceased to help, the patient was worse till a new source of aid was forthcoming. And as one does not consult a doctor unless one is ill, the illness aspect of the problem in those who have come to rely on doctors, in those therefore whom we doctors see, tends to become accentuated; and the aspect which has to deal with difficulties other than those connected with health tends to disappear from the history. Then illness comes to be exploited. It not only brings support but gets one out of difficulties; for one cannot be expected to face them when one is ill.

From what has been said it might be inferred that the difficulties which beset the patient begin in childhood and it is probable that they always do so;

with ordinary history-taking—and that is all we are discussing at present—this will commonly be found true, but not always. Sometimes childhood appears to have been largely forgotten; it is not forgotten if the patient be kept to the subject for several sittings, but for the purpose of diagnosis at a single interview it may be. But when this history can be obtained it will be found that childhood was unfortunate in one of several directions; the child was spoiled and acquired the habit of dominance which later the man cannot exercise, or was fussed over and so feared this world too much, or was unduly snubbed, or was treated with harshness and became terrified, or was mismanaged in some other way.

A person who gives a history of this sort is a neuropath; he may of course be suffering from organic disease also, but whatever he is suffering from he is the type of person who will require psychotherapy in addition to whatever physiotherapy is indicated.

Before examples are given of psychogenic histories it might be well to give some more which might deceive the unwary.

Case 2. M., age 50. He had a history of varicose veins in the left leg for years. Six months before coming under observation he had suffered from pain in this limb, said to be due to phlebitis; it was better in two weeks. A few days later he had pain in the back and hypogastrium, and soon after pain in the right calf. He began to be constipated, having previously been regular. The constipation became severe, five or six days being missed. The pain in the abdomen increased. He was sent to a nursing-home for a week's investigation, and was carefully examined by many modern methods. Nothing was found and he was stated to be suffering from functional disease, and was further said to be giving daily proof that this was so. There was no history of previous ill-health, and none of increasing difficulties in his biography. Doubt was therefore expressed about the diagnosis, but the following story was put forward to prove the functional nature of the case.

It appeared that his doctor, when visiting him one day, had observed him through the window reading the paper, and apparently sitting at his ease. A minute later on coming into his room he found the patient writhing on his bed in great pain.

We may agree that this was an exhibition of malingering: we may sympathise with the temptation to label the case as hysterical when this observation was combined with absence of physical signs, and yet if we consider the matter a little further, the incident may have an explanation which it might be difficult for a doctor to appreciate, because it was a rather pungent criticism on doctors in general. Malingering is not a very common mode of reaction. It is practised only by very helpless people in the face of what is to them an overwhelming difficulty. A man must feel himself in a very tight corner before he resorts to it. If we diagnose malingering our first question should be, what is the difficulty from which this person sees no way of escape? This man was involved in no external difficulty. He had only one difficulty at all; he was in great pain and no one would believe it. He was making a poor attempt to prove his case. I think that many of us faced with the denial that we were in great pain, when we knew that we were feeling it, would take steps to assure our informant that he was wrong. There are three great

motives for malingering—getting money for illness, escaping loathsome duty evading punishment; and this man had none of them. He was reacting neuropathically, but this was the only time in his history that such a thing had been shown to have occurred, and everyone reacts emotionally, hysterically, deceitfully, if you like, sometime in his life. It is not normal not to do so ever.

A few weeks later he became jaundiced, and was found to be suffering from cancer of the pancreas.

CASE 3. F., age 27. In 1921 the patient suffered from loss of power in arms and legs, attacks of shortness of breath, sleeplessness. In 1917 a brother was killed; in 1920 her house in Ireland was raided; her father was wounded before her eyes, and she rode ten miles in the night for aid. Later that year she began to sleep badly, came to England and recovered. She returned to Ireland and went on normally. The house was burnt down three months later, but there was no shooting. The sleeplessness returned and she developed the symptoms as above. The illness was regarded as hysterical. There was no previous history of ill-health or of increasing disability to meet the problems of life.

About eighteen months later she was definitely a Parkinsonian.

The temptation to regard a history of this sort as psychogenic is of course very great. There is abundant material for the production of shock, but it is probable that shock in itself does not produce nervous illness, not even shell-shock. If one takes the history of men invalided on account of shell-shock, one will find the story of months of terror preceding the shock. There was no clear history at all that this lady had terror at any part of the proceedings. Her ride in the night for help for her wounded father was indeed proof, so far as it goes, to the contrary. Though she came to England she went back to the front. Did any shell-shock patients who were invalided to England return to the French front? Some were sent back, but it seems probable that none stayed more than a day or two. She on the other hand went back voluntarily and stayed, until there was no house to stay in. The first attack of sleeplessness may have been an anxiety reaction; it disappeared quickly on removal of the disturbing cause. A certain amount of sleeplessness in the presence of anxiety need not be regarded as illness; poor sleep is an ordinary accompaniment of normal anxiety; it is only its prolongation after removal of the precipitating cause that need be considered pathological. The second attack of sleeplessness was probably encephalitic. There was no previous history of inability to meet difficulty.

The next case to be considered has a still more provocative history. It is the sort of history which might be called neurotic if the performance of certain ill-judged and unworthy acts is to be the criterion. Only those conscious of their own invariable wisdom and rectitude will, however, be inclined to say: "that is the sort of conduct that leads to neurosis." The point is not what one does, but how one reacts emotionally to what one does, otherwise the criminal and the psychoneurotic would be the same person.

CASE 4. M, age 38. The patient had been well and active till two and a half years before Eight years previously he had married a widow ten years older than himself—the ill-judged act; and later had had sexual relations with his step-daughter—the unworthy

one. But he had remained well. Two and a half years before coming under observation he had a sleepless night, and next day had neuritis of the face and wrists. "His mind became a cesspool of all the sexual filth he had ever heard." Two days later he had an attack of vomiting which lasted seventeen days. This was followed by retching and eructations. In three months, though still retching and eructating, he returned to work, but these symptoms continued to be so severe that he had to give up. It was considered possible that they were due to a hernia which had developed in an appendicectomy scar. This was operated on with benefit so far as the vomiting was concerned, but not in respect of the eructations. On coming under observation, the patient was exceedingly depressed, confessed his sins without pressure, said that his illness was brought about by them, and that he was unworthy to live. He was then in a well-advanced state of Parkinsonian rigidity.

Now the ready confession of sins is no part of a psychoneurotic history. The psychoneurotic does not confess his sins; not till he knows where he stands with the particular doctor: on the contrary he tends rather to blame other people for the troubles with which he is being overwhelmed. There is nothing here, then, of a history which comes into the category as laid down in an earlier part of this paper; there had been no disability to meet difficult situations till he had actually fallen ill.

So far as physical disease is concerned we have endeavoured to show that there is, in so far as the patient is suffering from the physical disease alone, an absence of a previous history of increasing disability to meet the difficulties of life. In the absence of such history little stress need be laid on trauma, physical or mental, except as a mere precipitating agent, or on the observation that the patient is not reacting courageously to pain. It will be of interest to see whether the biographical method will be of equal service in enabling us to distinguish between the psychoneuroses and two great groups of psychotic disorders with which they are liable to be confused—the depressions and the schizophrenias. I am not entering the lists on either side on the question whether these psychoses are psychogenic or physicogenic. I am interested to prove that the psychoneuroses are psychogenic, and that they are different conditions from these psychoses, not in degree but in kind. This paper will not afford sufficient proof to dogmatise on the latter question, but it may provide enough to show that if these conditions are psychogenic the histories are not of the kind postulated for the psychoneuroses. In their full-blown state they affect quite different functions of the mind. If then they are psychogenic one should expect a different story about their psychogeny.

If the cross-section method of diagnosis be employed there are stages where the resemblance between these psychoses and the psychoneuroses is considerable, and therefore we must resort once more to the biographical method.

Case 5. M., age 51. On coming under observation the patient said he was worried about his financial position—on information received, unnecessarily so. He slept badly and could not read because of inability to concentrate. He spoke slowly and deliberately and did not answer at once. He was greatly constipated, and had lost weight. He accounted for his illness by saying that he had been slack and inattentive to business.

(The above is a cross-section diagram of an attack of psychotic depression.)

Eight months previously he had been perfectly well. About that time his only son went abroad, to be away for a long time. He received news that a brother, also abroad, was in serious trouble and this was a great shock. He thought that the two events might have preyed on his mind. He soon began to lose interest in both work and games, and the other symptoms came on gradually. Two months after coming under observation he was well.

When he was better, he told me that during the illness the thought of suicide had often come into his mind, but that some other thought had always driven it out. The story of the attack corresponds therefore to the cross-section diagram. We may consider that the affair of the brother was a precipitating event.

The longer history showed that there had been two similar attacks years apart, each lasting about six months. But between them the patient had been perfectly well. There had been these incidental illnesses, in which there had been complete inability to do anything, rapidly arrived at, and finished in a few months. When he was not ill in this way he was always a singularly effective and successful man. His life had been spent in colonial administration and he had climbed to the top of the tree. His wife described him as energetic, self-reliant, masterful. There is here no picture such as has been laid down as essential to the conception of a psychogenic history. Indeed the biography shows one of quite an opposite nature. When he was well, he was never troubled with nervous doubts or fears; he had no doubts about himself.

The schizophrenic person on the other hand tends to furnish a history which may show pathological happenings early in life and they may be somewhat like those characteristic of the psychoneurotic. Yet no such story may be forthcoming. It will be convenient to give an example of the second kind first.

Case 6. F., age 29. She came under observation in 1924. Her complaints were like those of a patient suffering from an anxiety state: they were occasional headache, fatigue, nervousness in company, starting on going to sleep, poor sleep if worried, eructations. On examination nothing was found to suggest anything but the presence of a psychoneurosis, except that she was very listless.

At the first interview the psychoneurotic is not usually listless. If she says she is exhausted she often appears to let the doctor see it in a very thorough fashion—she will be deep in the bed, hardly able to articulate, but more usually the first meeting with such a patient is a pleasant social affair. But this lady gave one the impression that she was quite uninterested.

Her recent history was somewhat suggestive of an anxiety state. Her mother had been ill for some years with paralysis agitans; after an air raid the patient had become nervous, depressed, could not sleep well. She left town and was well in three weeks. She remained well and at work till about a year before she came under care. At that time she had an attack of chickenpox. Since then though she improved a little she never became quite well, and of late her symptoms, chiefly those of fatigue, had become worse. Previously to the air raid she never showed any signs of finding life difficult.

In hospital all she wanted to do was to lie in bed. She would talk when spoken to and answer intelligently, but she had little to say, and nothing of much importance to relate. When she had been six weeks under observation she confided one day that when she was in Switzerland convalescing from chickenpox she had been lying in bed one night looking out on to a balcony, when suddenly a panther appeared outside the window and stared in at her. She did not call for help, but next morning she saw the marks of its pads on the matting of the corridor outside her door. A few days later while menstruating she said she was much troubled because she was about four months pregnant. When asked if she knew that this state of affairs was hardly compatible with her menstrual regularity, she ignored the point, and proceeded with her confidences, which were that even in hospital she was nightly visited by a lover whom she had never seen, for he always came in the dark, but whom she had always been able to touch. This fantasy persisted, and soon after she was removed from my care. I have ascertained, however, that the case proceeded to certification and that the patient is still, four years later, in a mental hospital.

Both in cross and longitudinal section this case differs from the anxiety state. Apathy is not a characteristic of the latter condition. No history of abnormality was discovered in the early years.

Case 7. M., age 20. When the patient came under observation his condition was frankly schizophrenic, with auditory hallucinations and ideas of persecution. His history was not that of a normal person, but it was not exactly that of one who had often been overcome by difficulties. He was stated to have been always nervous; on closer inquiry what this meant was that he had never made friends easily, that he was always a silent person who had kept himself to himself. He had done quite well with work. He had been introverted.

There is no real history here of increasing difficulties till he fell ill; but this is the sort of history which is a little difficult to distinguish from that of the psychoneurotic, and schizophrenic patients are the most difficult to distinguish from the neurotic in the earlier phases of the disease.

We are now in a position to consider the history of some patients with anxiety states. We shall begin with one where the diagnosis of a functional nervous disorder and also of a physical illness is clear on cross section, and observe how the history will enable us to distinguish between the physical and psychic elements.

Case 8. The patient, a childless married woman of 46, complained of utter exhaustion and of attacks during which as a rule she did not lose consciousness, though sometimes she did. She became stiff, the neck jerked, then the head and neck turned to the right, sometimes the whole body did so too; the arms were stiffly extended and pronated; the legs were stiff and extended. Sometimes the arms then went up in a crucifixion attitude; both legs might swing out to the left. She had sometimes fallen out of bed; she had had opisthotonos. The attacks lasted a few minutes, but one had continued for at least twenty minutes. She had sometimes been held down by nurses and then the attacks lasted much longer. They had been present off and on for years. She complained also of indigestion. There was complaint further of œdema of the legs on both sides when she was especially over-tired.

A day or two after she came under observation I had the opportunity of seeing an attack and was able to confirm—what her own description had suggested—that they were of hysterical nature.

The patient was asked when the first attack occurred and she said that it was seventeen years before, when she was 29 years of age. She had come home on leave from Canada

where she was employed as a schoolmistress. One day a favourite uncle had called. She thought he looked very ill, but was unable to persuade her mother and sister that he was. A few days later he was dead; and a few days after that she had the fit.

It may seem here that stress is being laid on the occurrence of what it was promised that stress should not be laid on, viz., the presence of shock in the history; but it may be noted that she had no more of these fits for several years, and it was only within the last two that they had reappeared. She did not therefore become hysteric in consequence of this shock, but, as happens to all hysterics, she reacted more violently to shock than does the ordinary person. We shall find evidence of hysteria long before this event happened. We shall find also that this particular shock had a special signification.

As a child she was not happy; her father terrified them all; her mother made a favourite of a younger sister. As a quite young child she remembered having frequent abdominal pains. For these she would steal out of bed and stand in front of the kitchen fire and warm her abdomen, which comforted them. She would get whipped for doing so if caught. She remembers the agony of the knee over which she was turned pressing into the abdomen. At the same period she was terrified of the dark, because she sometimes had a visual hallucination of a white coffin being carried out of her own bedroom.

Here we have the recollection of physical disease—appendicitis probably, in the light of subsequent history—treated by methods of terror, so that a neurosis (hysterical hallucination) was induced. The organic indeed caused the functional, not by toxines or even by physical pain, but through the psychological route of fear. Here too is the genesis of something which pervaded her history from childhood onwards, that whenever she was ill she had a feeling of guilt, that as she was punished for being ill, it must be her fault. The reasoning is illogical, but children are often illogical. A number of other ideas besides those of guilt seemed to result from this treatment also, and it need not surprise us if these were incompatible with it. One of them was a feeling of injustice. That a person may feel guilty and at the same time feel that the treatment meted out is unjust is not remarkable. We all tend to feel that way; one might put it that one part of us accepts the punishment and another part resents it. And if we have felt injustice frequently we shall soon see injustice in places where it has not really been dealt to us.

It came about therefore that this patient began to think that her somewhat younger sister was receiving far more benefits than she was; it seemed to her that their mother was always heaping good things on her, and she therefore became jealous and resentful towards her. She soon got the reputation of being sullen and bad-tempered, and as this was in marked contrast to the sweet gentleness of the sister, it probably became true sooner or later that the latter did in fact get more privileges than she did; for this was one of these Scottish households where the naughty child is not allowed to rule, but is perpetually and unweariedly put in her place. We can now see how this child was almost bound to go on making bad adaptations, and we may learn by the way how difficult it may be to solve the problem of the naughty child unless the whole family will submit to a searching interrogation.

The patient therefore became a misfit at home, and as soon as she grew up she was glad to take a post overseas. When she came home for her first holiday the death of the uncle occurred which was followed by the fit. While she was abroad she had had endless difficulties with her employers, who had constantly overworked her; she had had several love affairs, all with wrong persons—one of them, for example, was sent to jail for swindling.

A series of unfortunate love affairs must I think be regarded as something more than accidental. It argues bad management. In this case I consider it signifies that the patient was so desirous of matrimony that she would think of it in connection with anyone rather than not have it. And for her the reason for the desire of matrimony was not hard to seek. It does not seem as if she had had any very strong sex urges. In the history there was nothing to suggest them. But she had a passionate desire for certain things she had never had—to be cared for and to be supported. The instinct that was threatened was probably more that of self-preservation than that of sex.

At the same time there were frequent attacks of abdominal pain and other abdominal symptoms so that when she came home a second time in 1914 it was decided to open the abdomen. An appendix buried in dense old adhesions was discovered and dealt with. The operation was a grave one, was followed by phlebitis of both limbs with permanent double varix. These adhesions had been the source of much delay in the passage of the intestinal contents—this appeared to have been benefited by the operation. In later years, at any rate, there was no stasis.

Here, then, we have sufficient proof of the organic nature of the childhood pains, and the foundation of the subsequent œdema of the legs.

We begin to perceive how history aids us in disentangling the organic from the psychogenic. We see why the child became ill-adapted and why she was likely to find the ordinary difficulties of life greater than most people do. We see also why she had organic symptoms. Of course no one is likely to think of œdema as functional; but in a complex case like this it is essential that the patient should be able to distinguish between what parts are in the one category and what parts are in the other. She may or may not accept the doctor's statement on the matter; this patient was not apt in doing so, for she had had many opinions from many doctors, and she had almost given up believing in any; but as she assisted in the dissections of her symptoms she began to see for herself that they were in two categories, and that it was not very hard to make a distinction. Thus she knew that the hallucination of coffins was not likely to be caused by appendicitis, for she had known other people with that disease, and they did not suffer from hallucinations. She could see that prolonged jealousy was likely to be followed by symptoms of some kind, and she could see that the death of an uncle was not likely to cause an organic affection. While making these considerations she was able to supply a reason why the death of this particular person had affected her so much. He it was who administered the corporal punishment referred to in her childhood, and she had often wished him dead: later he had been kind to her and she had liked him, and had perhaps been unhappy in the knowledge that she had wished him dead. This is however a psychoanalytic hypothesis

and not a historical fact, but it is not very far-fetched to postulate that when anyone against whom we have had death wishes at one time and yet liked at another time, does die, we shall feel specially upset about it.

> Time went on and at the age of 40 she married; and marriage failed to give her all she wanted. It gave her money, it gave her leisure, but it did not give her what she had craved for all her life, viz., the gratification of her desire to obtain attention and affection from somebody. Her husband was quite kind but he was very busy, and gave her very little attention, and if there is no attention affection is not worth much. They lived in the East and had innumerable servants who left her nothing to do; they came home and for the first time in her life she tried to manage a large London house; she came to grief, and retired to bed exhausted. Unfortunately massage was prescribed; and soon she had phlebitis once more in the old damaged veins. This was followed by pulmonary embolism and she had a fight for life. In this fight the fits recurred.

She knew she might die. Death had always been a source of terror. The hallucination of the white coffin in childhood—white for a child, white for herself, therefore—may be adduced in support. The first fit had occurred in connection with a death. It is of some interest that for some weeks the fits occurred only in the presence of the doctor. It might be natural to suppose that here we have a desire to gain the doctor's attention; that would accord with the ordinary banal view of hysteria; but the patient maintained that she had by this time got so terrified of doctors that this one appeared to her as the minister of death. She had herself associated the onset of the phlebitis with the massage; the rubbing had hurt the veins; then they were declared inflamed; then she had become more ill with the embolus than she had ever felt in her life. The doctor had been the agent in bringing all this about.

In the next two years there had been apparently considerable doubt about the nature of these attacks. They had been considered epileptic; if not epileptic they had been considered toxic, because she had had attacks of coli pyelitis; fancy osteopathic diagnoses had been gone in for. No doctor after the first one had seen an attack. After she had left him she had not had them in the presence of doctors. The description seemed clear enough; but the history would have decided the matter.

Now the proof that these fits were psychogenic lies in the fact that they disappeared when her mind was relieved, which happened after this history had been gone into. She had been called "functional"; she had been called "organic." It had not presented itself to her mind that she was clearly both, and that separately; the one did not depend on the other, except through the channel of the mind. The organic diseases which she had were admitted, and shown not to be fatal. The mental cravings and self-reproaches were put by her in their proper light after she had discussed them.

It must be admitted that this history was not obtained in one or two interviews; it took many, and it will be objected that no one can find time to obtain a history of this kind. In a diagnostic clinic, by the time a patient has had all the routine examinations (chemical, bacteriological, radiological, etc.), the aggregate number of hours consumed must be very great, quite as

great as that consumed in the thorough taking of a history in a doubtful case, and from the latter investigation the therapeutic advantage is sometimes greater than from the former. The trouble is that one man must do the latter alone without assistants; in the former the chief waves a wand and an army of helpers does the rest; and nobody becomes tired. But a diagnostic team for doubtful cases should include someone who could be taking history, while other people are developing their films and staining their slides. In ordinary practice such lengthy procedures are not often called for, and even in doubtful cases sufficient indications that neurosis is present—which, after the example just given, is not to be confused with "organic disease absent"—will often be found at a single sitting.

CASE 9. A single woman of 22 complained of aching in the eyes, worse on reading, so that she had almost given up reading altogether, and of headaches. The eyes often became red and inflamed. Her own doctor said she had strained her eyes, prescribed glasses and said the pains would soon be better; they became worse. She went to an ophthalmic hospital where they gave her glasses (but said she need not wear them much), an eye-wash and an ointment. There was almost immediately complete recovery from the symptoms. A few months later the headaches and the ocular symptoms returned suddenly. The headaches became so bad that her doctor sent her to bed for six weeks. There was no improvement. Then she was sent to a general teaching hospital where she was given further rest, her eyes painted with a silver preparation, and irrigated night and morning. No improvement followed. She then saw the ophthalmologist to the hospital, who said there was nothing wrong with the eyes.

This in itself is a neurotic history. The first ophthalmologist evidently did not think there was much wrong or he would not have ordered only occasional use of the glasses. The second said there was nothing. We are entitled to suppose that the first one corrected a normal astigmatism; his doing so would give the patient some hope, the greatest of all the methods of temporarily improving neurosis. It is clear however that the patient reacted in a faulty way. She did not react well to her own doctor who was "not a specialist"; she did react to the special hospital: she reacted favourably to prestige. Consequently when the doctor, who had failed once, tried to improve her by putting her to bed he failed again. The whole series of reactions is faulty. However, if this is not enough, a very cursory inquiry into the history reveals that the relapse coincided with a grave annoyance which had happened. The patient had contrived to get a sister a post in the business in which she worked herself; and this sister was very quickly promoted over her head, and she did not like it. Therefore the relapse also was a faulty reaction. Her jealousy was intensified because she knew nobody but her sisters with any degree of intimacy; she had no outlet outside the family. There were four sisters. They never went to other people's houses and no one came to theirs. This had always been so. And this represents a mass faulty reaction which this family had made. The father had been a drunkard, and had attempted suicide long before the patient was old enough to know anything about it; the mother became very sensitive and had told the girls about it; and they all kept themselves to themselves.

This history as stated above was all obtained in one sitting. It is free from any great shocks or great strains. It is clearly however a history of faulty methods.

In the first of these two histories (Case 8) the psychogenic part is easily separable from the physicogenic by cross-section methods. The interest in the detail of the case lies in this, that the history shows that mental factors clearly accounted for what is called hysteria, physical factors for organic symptoms like œdema. The second case (Case 9) is of interest because it is of the kind which are often quoted as proof that physical changes are the important thing in functional disease. The important physical thing was a slight astigmatism, evidently one not more than that which occurs in most people; and the subsequent events exemplify what often happens when normal astigmatism is successfully exploited in the treatment of neurosis. There is immediate improvement, and then relapse at the first breath of misfortune. It is no argument against a psychical view of the neurosis if such relapse occurs after psychotherapy; but relapse after successful physical therapy is wholly damnatory to the physical view. One's sister getting a better post than oneself could not alter one's astigmatism; it might alter self-confidence even if that had been temporarily restored by mental means.

The first patient we see is easily recognised as a probable neuropath, the second as a likely one by cross-section examination. We must study finally a patient where the diagnosis was not obvious or simple, and where the history at first sight does not seem to yield the series of faulty adaptations which are imperative if the thesis of this paper is to be upheld.

Case 10. M., age 53. The patient complained of slight tremor of the left arm and leg, headache worse after greasy food, buzzing in the ears, concentration not so good as it was, but not preventing him from going on with his work, and nervousness. These symptoms had been present for about two years. He had never been a good sleeper. He had had an illness towards the end of the war characterised chiefly by exhaustion; without doubt he had worked unusually hard during the war. Since then he had had a good many minor manifestations of ill-health at times, with symptoms of dyspepsia, shivering attacks without much temperature. He had been operated on for a duodenal ulcer which was not there, and the surgeon had thereupon removed the appendix and gall-bladder, but the dyspeptic tendencies had remained.

(All this story is explicable on an organic basis. There might have been toxæmia; latterly there might have been encephalitis.)

The man himself was a highly successful person. At about the age of 20 he had quarrelled with his father. When he did this he was thrown wholly on his own resources. He became a schoolmaster, then supported himself by journalism, was called to the bar, took up business and was made director in an undertaking of great importance, a post he now holds; later on he took up public affairs and has been highly successful.

Here then seems to be a man who gives the lie direct to the conception of the neurotic individual whose fancy portrait was painted at the opening of this paper. Let us see. It was never said here that the neurotic lacked courage, or ability, or energy. It was said that he made more faulty reactions than most of us, that he came to rely on something, and that he came to exploit

illness. Many neurotics have marked ability, but in important ways they seem to have lost the art of living. This man's ability helped him to overcome many things, but there was much in his history that suggested faulty reactions in excess. As a child he could not be left alone. He had not got on badly with his father in youth, and then he quarrelled so hopelessly with him that he refused to take further help from him; the father wanted him to understand that he expected him to pursue a certain career when his education was finished; and as he felt he could not follow his wishes he would accept nothing more from him. It is very magnificent, but it is questionable if it was healthy. Later he was twice engaged to be married. The first engagement was given up on financial grounds, the second because of incompatibility. After that he seemed to lose interest in the subject. There is more than one faulty reaction here. The cost of marriage if it is to be counted at all should be counted before engagement, not after. The normal person does not usually count it very much; the dropping of the subject argues a failure of a normal instinctive 'drive.'

Up to this point the reactions had not resulted in symptoms of illness, but the illness at the end of the war was a suspicious one; certainly after it he never ceased to consult doctors and undoubtedly there was exploitation. During the war he had intense misery because he had not been permitted to join the army. Both his private and public work was too important for the authorities to allow him to do so. He felt ashamed and disgraced. This may be seen as a development of that early over-scrupulousness which had inhibited him from accepting further parental assistance. When the war was over he was often disgusted at the sordid nature of public work, and once he retired from it for a year, not sorry that he was too ill to attend to it. He had returned to it from a sense of duty, but when he came under care he was heartily sick of it, and at the same time not well enough to do himself justice at it.

Here then we have a man with a perfectly conscious conflict which he felt unable to solve, but which illness from time to time shelved, a man, who, as soon as we have penetrated his superficialities, presents us with exactly the psychical characteristics which were laid down as those we might expect to find in the history of a neuropath. Here amidst seeming success and self-reliance we have increasing dependence on doctors and exploitation of illness. And it must be emphasised that neurotic manifestations began long before there were likely to be infective causes, and that they continued after likely causes were abolished; nay more, they grew worse. It is easier to explain events like this on mental than on physical grounds and though it is easier that does not mean that the explanation is facile. It is difficult to conceive of the toxæmia that could have made him over-scrupulous for thirty years, and yet left everyone unable to find where it was at the end of that time.

The study of these cases seems to demonstrate that there is such a thing as a psychogenic history, that illness does seem to be definitely correlated with mental states, and that these states are of very early origin. They show that

shocks and strains are not the essential points of such a history. Their importance lies only in the fact that shocks provide a time of testing for the weaker vessel, so that they seem to the casual observer to be of greater importance than they are. The physicogenist will say that even if such histories are true, and if we can recognise them—for it does require practice to use the instrument of biography as it does to use any other special instrument—then we have got back to the physical after all; for what kind of person we are is probably determined chemically. This cannot be wholly true. If it were true it would not matter in the least how we were brought up, and all the thought expended on education in the broadest sense would be wasted. It is moreover true that psychotherapy does sometimes seem to make people well who were chronic invalids before it was administered. We all admit that there are endocrine changes in neuropaths, but this is not to admit that therefore endocrine therapy is the best. We knew that the blood-vessels of the skin dilated easily in neuropathic persons long before internal secretions were thought of; we knew, that is to say, that mental changes coincided in time with physical changes and that the changes were correlated. We even knew that these people sometimes said that their blushing made them nervous; but it was often easy to show them that it was first of all their nervousness that made them blush, though the blushing increased the nervousness in its turn. It is nothing new, then, to know that there are definite physical changes in mental states. The question remains open—which came first? and it is unanswerable. But it is probable that at our present stage the mental attack is the more likely to succeed, and it is likely that often when we think we are attacking physically we are doing so mentally. The mental is the more difficult form of attack. It often becomes incredibly tedious to both physician and patient; and one cannot help wondering whether this may not be one of the reasons for its periodical partial eclipse.

A STUDY OF MUSCLE IN CONTRACTURE: THE PERMANENT SHORTENING OF MUSCLES CAUSED BY TENOTOMY AND TETANUS TOXIN.

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INTRODUCTION AND DEFINITIONS.

The term contracture is used to designate a variety of conditions of more or less permanent muscle shortening. Some of these need only be mentioned. The delayed relaxation seen in a nerve-muscle preparation after a series of rapidly repeated contractions, the delayed relaxation caused by veratrine poisoning, the prolonged shortening caused by nicotine and acetylcholine in frog muscle and in denervated mammalian muscle, and Tigel's contracture are chiefly of interest to the physiologist. With the exception of the first-mentioned, which is generally, though perhaps mistakenly, regarded as a fatigue contracture due to the accumulation of lactic acid in the muscle, not enough is known about the physico-chemical factors involved in these phenomena to make them of any value in the solution of the problems to be considered in this paper. Nor are the structural factors any better understood. The relative parts played by the myofibrils and sarcoplasm are still matters of controversy. It is not known whether or not nicotine and acetylcholine produce contracture by stimulating a special receptive substance in the muscle associated with hypothetical parasympathetic endings, as maintained by Riesser and Neuschloss[1] and Frank, Nothmann and Hirsch-Kauffmann[2]. Certainly this conception has received no support from the recent work of Gasser and Dale[3].

As ordinarily used in clinical literature, contracture designates a condition of persistent tonic shortening of muscles which will, however, relax during sleep or under anæsthesia. To this class belong the contractures seen in some patients with lesions of the pyramidal tract. This sort of muscle-shortening is maintained by a continuous stream of nerve impulses reaching the muscles from the central nervous system and affecting one group of muscles predominantly or to the complete exclusion of their antagonists. It is, in fact, nothing but an exaggerated and continuous state of hypertonus and is characterized by the deformity which it produces, by its steady persistence during the waking hours, and by the fact that it disappears during deep sleep or anæsthesia[4]. Hysterical contracture and the reflex contractures, so often seen during the war, which bear some obscure relation to more or less trifling wounds, also have as

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their basis an excessive and continuous tonic innervation of the affected muscles. Muscle-shortenings of this sort, whatever their cause, fall in one great group and may properly be designated as hypertonic contractures.

But apparently even in contractures of pyramidal origin there may develop a certain amount of intrinsic shortening in the muscles, for according to Foerster[5] while these contractures are due in large part to hypertonus, they are also in part due to the fact that each muscle group adapts itself to the approximating of its points of insertion by the gradual development of contraction and permanently retains this condition of shortening. If this actually occurs, the affected muscles should not fully relax under an anæsthetic and the residual shortening that persisted under deep anæsthesia would belong in the class to be described next.

Under the general heading of myostatic contractures we propose to include a group quite different from the preceding. It includes all contractures that are caused by the fixation of the muscle at a given length for a considerable period of time. Familiar examples are the contractures which restrict the movements of joints after immobilization for weeks in plaster casts, the permanent shortening of muscles after division of their tendons, and probably, in their early stages at least, the paretic contractures due to the unequal paralysis of antagonistic muscle groups in anterior poliomyelitis and multiple neuritis. This group, because they all are caused by fixation of the muscle for a long time at one unchanging length, might not improperly be designated as immobilization-contractures, but we feel justified in using the word myostatic as more accurately descriptive of the condition.

By myostatic contracture we mean to designate a condition of permanent shortening in resting muscle which is maintained in the entire absence of nerve impulses, the muscle having acquired, usually as a result of prolonged immobility, a new and shorter resting length. It is the condition which has been designated "Ruheversteifung" by Spiegel and Shiboya[6]. These investigators and others as far back as 1886 (Moll[7]) have clearly demonstrated that if the bony attachments of a skeletal muscle are immobilized for several days so as to prevent the changes in length which normally result from spontaneous and reflex movements, there occurs a fixation of the muscle at the length thus imposed upon it. If taken sufficiently early, this contracture can be overcome by active or passive movements, but if it is left untreated for some time the damage becomes irreparable.

It may be that adhesions and ankylosis play a part in the limitation of motion in a joint that has been enclosed for weeks in a plaster cast, but these factors are certainly secondary to the shortening of the muscles. This is clearly shown by the following experiment (Moll[7]). After the hind leg of a rabbit had been enclosed in a plaster cast for 12 days, division of the muscles completely eliminated all of the resistance to passive movement which had developed. This showed that within this time no adhesions, thickenings of the joint capsule or changes in the articular surfaces had developed and that the limitation of movement was entirely due to a shortening and fixation of the muscles.

Fröhlich and Meyer[8] have shown that this fixation of an immobilized muscle is dependent upon the nerve impulses which reach it from the central nervous system. Immobilization of a denervated muscle does not cause it to become set or alter its extensibility as would be the case if the nerve supply were intact. According to these authors section of the dorsal roots supplying the muscle is sufficient to prevent the contracture, indicating that the integrity of the local reflex arc is essential for its development.

Spiegel and Shiboya[6] removed the left motor cortex from cats and found that in the right hind leg the extensor muscles became set more quickly than the flexors, while flexor rigidity was more easily induced on the left side. They carried out other experiments, transecting the spinal cord in the lower thoracic region and then putting the limbs up in plaster casts in the flexed position, and found that setting occurred in the fore limb more quickly than in the hind limb. These experiments indicate that the brain centres which control tonic innervation of the muscles play an important part in the genesis of this phenomenon.

Myostatic contracture is then the fixation or setting of an immobilized muscle in a new and shorter than normal resting length. In its genesis it is dependent on the nervous system and it is probably an abnormal manifestation of tonic innervation. It is, however, only in its genesis that myostatic contracture is dependent on the nervous system. After it has once developed, it is independent of the innervation and persists under deep anæsthesia and even after section of the motor nerve. It is a contracture of muscle at rest and is in this respect to be sharply distinguished from the hypertonic contractures previously mentioned.

It is surprising that practically nothing is known concerning the chemical, physiological, or structural alterations in muscle which are responsible for the various types of contracture. Attempts at an explanation have dealt almost exclusively with the hypertonic forms and have been for the most part only reviews of the literature on the controversial questions of muscle tonus. The papers of Buscaino[9], Cooper[10], Crocq[11], Noica[12], Piéron[13] and Ott[14] contribute nothing of importance to the solution of the problem. Probably the most significant single observation is that attributed by Oppenheim[1] to Brissaud, namely, that an anæmia of the affected extremity produced by an Esmarch bandage has a relaxing effect on the shortened muscles. It suggests that the shortened state is not maintained by structural alterations in the muscle, but by a chemicophysical equilibrium that is upset by the anæmia.

We shall be concerned in this paper with the experimental myostatic contractures which can be produced by tenotomy or by tetanus toxin. After section of its tendon a muscle shortens. It is freed from the frequent stretching which normally results from the contraction of its antagonists. Under these conditions it undergoes the same changes in extensibility which occur in the muscles of a limb rigidly fixed in a plaster cast. It is what Spiegel and Shiboya have called "Ruheversteifung," as already remarked. Dixon and I[15] found

that 15 days after section of the tendo achillis the gastrocnemius muscle of the white rat had not only undergone shortening but it had become set at this new length and was less extensible than normal. This was due to a change in the muscle itself, because it persisted after section of the sciatic nerve. But in its genesis this contracture was dependent on nerve impulses, because it did not occur if the sciatic nerve was cut at the same time as the tendon. These experiments may be briefly summarized as follows.

In the white rat the tendo achillis was cut in both legs. The right sciatic nerve was also cut at the same time. The left sciatic nerve was cut at the time of the experiment, which was performed 15 days after the original operation. By that time the left gastrocnemius muscle, whose nerve had been intact up to the time of the experiment, was shortened, plump and firm. On the right side where the nerve had been cut at the same time as the tendon the muscle was of normal length, but was soft and flabby. While the circulation in these muscles was still intact they were stretched in an identical manner and for the same periods of time with loads increasing by ten gram intervals up to 100 grams. This method of stretching caused a permanent elongation of 2·82 mm. in the right gastrocnemius whose nerve had been cut at the same time as the tendon. This figure represents an average of 6 experiments. But in the left gastrocnemius whose nerve was intact until just before the stretching began the same weights acting for the same periods of time caused in the 10 experiments an average permanent elongation of 1·34 mm. Ten normal gastrocnemii from other rats gave, when stretched in the same way, a permanent elongation of 3·08 mm. This permanent elongation must not be confused with elastic stretch which disappears immediately after the weight is removed. We found that the elastic stretch was not decreased in the muscles after tenotomy; but the permanent elongation or ductility, which corresponds to what Blix[16] called "Nachdehnung," and Langelaan[17] called "plasticity," was reduced by half.

Taking into consideration the fact that all muscles are under continuous tonic innervation it is not hard to see why a muscle should shorten after section of its tendon. But it is not at all easy to understand why it becomes set in this shortened length and has its extensibility reduced; nor do we understand why this reduction of extensibility affects only the permanent elongation and not the elasticity of the muscle. It is clear, however, that the cause is to be found in some change in the muscle fibres, because it persists after section of the motor nerve. It is equally clear that in its genesis this change in the muscle fibres is dependent on the nerve supply, because it does not occur if the nerve is divided at the same time as the tendon. Whatever may be the factors involved they are the same as those that produce the muscular rigidity which limits the use of joints after immobilization.

A very similar, if not identical, condition develops in the late stages of experimental local tetanus. When an appropriate dose of tetanus toxin is injected into one limb of a highly resistant animal like the cat or rat, the spasms

develop only in the injected leg. When this local tetanus is fully developed all the extensor muscles are in continuous tonic contraction. This is not interrupted by fatigue and persists for days without relaxation. The limb is held continuously in rigid extension ; it is in extensor contracture. In the early stages of local tetanus the limb relaxes when the animal is anæsthetized, indicating that the condition is one of hypertonic contracture and is maintained by the continuous discharge of nerve impulses into the affected muscles. But in from 5 to 7 days after the injection, if the dose has been an adequate one, the muscles become set in this posture of rigid extension and fail to relax under deep anæsthesia or even after section of the motor nerve or the death of the animal (Ranson and Morris[18]). There are then two stages of tetanus rigidity which are to be sharply distinguished : hypertonic contracture, which disappears after nerve section ; and myostatic contracture which, after it has once developed, is independent of the nervous system. Although these facts have been known since 1903 when Meyer and Ransom[19] published their studies on tetanus, no serious attempt has been made to analyse these phenomena or to ascertain their significance for neurology.

Dixon and I found that the ductility of the gastrocnemii in the myostatic contracture of local tetanus was reduced to an average value of 1·28 mm., which is a reduction of 60 per cent. from the average normal value of 3·08 mm. The tests were made in the same manner as in the experiments with tenotomy. As yet we have not been able to find a satisfactory explanation of this decrease in ductility ; but experiments looking toward an elucidation of this problem are now under way.

One thing is certain. Microscopical sections of muscle in contracture following tenotomy or the injection of tetanus toxin reveal no structural change which could account for the muscle having taken on a new and shorter resting length. There is no increase in connective tissue and very little change in the muscle fibres except that they have shortened. This change in the length of the fibres will be discussed in detail in a subsequent paragraph. The slight changes in microscopical appearance of the muscle fibres, namely a decrease in the clearness of the cross striations and, in some instances, especially late in tetanus contracture, an increase in the number of nuclei, do not account for the fixation of the muscle in a shortened length.

OBJECT AND METHODS OF STUDY.

The primary object of this investigation was to determine what change, if any, occurs in the function of a muscle in myostatic contracture. Is its capacity for shortening during contraction and its speed of relaxation altered from normal ? We have also studied the changes in weight and length of the muscle and in the length of the muscle fibres in tetanus contracture and in that following tenotomy. Cats and albino rats were used for the experiments.

A series of seven young adult rats were injected subcutaneously in the upper part of the right thigh with tetanus-toxin. The first one died from an

overdose; but kymograph records of the contractions of the gastrocnemii muscles were obtained from the other six. The rats varied somewhat in their susceptibility to the toxin, and, since it was desired to produce in each as nearly as possible the same stage of local tetanus, the dose and time necessarily varied. Rat T-2 received ·01 cc. of the toxin 5 days before the tracings were made; T-3, ·01 cc. 7 days before; T-4, ·01 cc. 4 days and ·005 cc. 3 days before; T-5, ·015 4 days before; T-6, ·015 cc. 6 days before; and T-7, ·01 cc. 8 days, ·005 7 days and ·005 2 days before the tracings were made. When a high grade local tetanus had developed in the right hind leg, the rat was anæsthetized with ether. If the affected leg relaxed the animal was allowed to recover from the anæsthetic and in some instances was given another dose of toxin as mentioned above. It was tested with ether again a day or two later. If there was very little relaxation of the injected leg under deep ether anæsthesia this was considered as evidence that the desired stage of contracture had developed and the experiment could proceed.

Before discontinuing the ether anæsthesia, 1 cc. of 5 per cent. chloral hydrate was injected intraperitoneally. The sciatic nerve was then exposed throughout its length in the thigh and was cut as close as possible to its point of exit from the pelvis. The tendo achillis was then cut close to the posterior tip of the calcaneus, after a thread had been securely fastened around the tendon. The gastrocnemius was freed of all fascial attachments, up to its two heads. The soleus was separated from the gastrocnemius and divided above its insertion into the common tendon. Damage to the vessels of the popliteal space and the branches supplying the gastrocnemius was avoided. The posterior surface of the middle third of the femur was exposed and a special clamp rigidly secured to the bone. During the dissection, the nerve and muscle were frequently moistened with Ringer's isotonic solution, at 37° C.

The animal was then placed on a support with the body resting on a sloping platform, and the prepared leg partially immersed in Ringer's solution, kept at 37° C. The femur was then rigidly held by the clamp, so that after adjustment was completed no movement was possible.

The heavy linen thread attached to the tendon was passed over a pulley and fastened to an approximately isotonic marking lever, having an initial tension of 28 grams in the first experiment and 50 grams in the other experiments. This tension was exerted by a long band of rubber and necessarily increased somewhat when the lever was raised by the contracting muscle.

This method of loading offered no serious obstacle to the shortening of the muscle and yet left the lever practically free from inertia. By means of an adjusting screw the initial tension could be removed between periods of stimulation and the muscle permitted to sink into the Ringer's solution, which was kept at 37° C., the level of the solution being so adjusted that the muscle dropped into the fluid when the tension was removed.

The stimuli were delivered through an inductorium with interruptions in the primary circuit at the rate of 50 per second. By means of a special

device run by clock-work and employing a platinum-mercury contact it was possible to close the primary circuit for a constant period of time. Nine or sometimes ten successive make and break shocks at a constant rate of 50 per second were obtained in this way from the secondary of the induction coil. A marker in the primary circuit indicated the number of stimuli and the length of stimulation on the kymograph.

The sciatic nerve was stimulated by means of platinum electrodes. For direct stimulation of the muscle one lead from the secondary was placed in the Ringer's solution and a second electrode of very fine wire was attached to the tendon. In this way the current had to pass down the entire length of the muscle. It was necessary to increase the intensity of the current because of the increased resistance of the solution and the fine wire, but no change was made in time or rate of stimulation. For indirect stimulation the secondary coil was usually set at 9 but for direct stimulation it was moved up to 5 or 6. The stimulus used in each case gave a just maximal stimulation, producing a typical tetanic contraction of the muscle. In some instances, where faulty contact between the nerve and electrodes occurred, an incomplete fusion was present. These curves were not included in the data presented. A time interval of two minutes was given between successive periods of stimulation to obviate any variable factor caused by fatigue. A tuning fork of 100 vibrations per second furnished the time record.

The right leg, which had received the injection or on which the tenotomy had been performed, was always tested first, in order that any ill effects due to poor circulation, cooling, prolonged anæsthesia, shock, etc., which might cause a decrease in the height of contraction or a slowing of relaxation, would show chiefly in the normal or control leg. Such deviations from the normal as were detected in the right leg must therefore be attributed to tetanus or tenotomy and not to any of the factors listed above.

Two cats with local tetanus were studied. One and one-half cc. of tetanus toxin was injected into the left quadriceps of one normal cat. After seven days the animal was prepared in the same manner as the rats with the exception that instead of fixing the femur by means of a clamp, a drill was passed through the femur, just above the origins of the gastrocnemius, and by means of a frame and cross-bars the femur and tibia were held rigidly in position. The leg was not immersed in Ringer's as in the rat series, but the muscle was prevented from drying by covering it with cotton saturated with Ringer's solution at 37° C. between periods of stimulation. A second cat was injected with one cc. of tetanus toxin in the left quadriceps. The next day the dorsal roots from the third lumbar to the third sacral were cut on the left side. After a period of eight days following the injection, the animal was prepared and tested in a manner identical with that of the first cat.

After the animals were tested they were killed, the gastrocnemii were excised along with the lower end of the femur to which they were left attached,

and fixed in 10 per cent. formalin. They were suspended with the femur as the upper support, and were kept under tension by a weight of 25 grams attached to the tendon until fixation of the muscle was complete.

In another series of experiments the tendo achillis on the right side was severed with aseptic precautions in six rats and three cats. After the lapse of from fifteen to twenty days following the tenotomy the animals were prepared and kymograph records of the contractions of the gastrocnemii obtained. The procedure used in the experiments was identical with that used in the tetanus series. The records were analysed in the same manner.

In order to determine the height of contraction and the rate of relaxation the kymograph records were analysed as follows.

The base line was prolonged below the curve and a perpendicular AC was dropped to the base line from A, the point of termination of the contraction plateau and the beginning of relaxation (Fig. 1). The measured length of AC indicates the height of contraction as recorded on the tracing. A line BE, parallel to the base line, was drawn through the perpendicular at a point equal to three-fourths the distance AC, measured from A, cutting the relaxation curve at D. A line DI, parallel to AC, was then drawn through D. AC was prolonged to H. The time in hundredths of a second between H and I represents the period required for three-fourths of the relaxation. It would be very difficult to measure the time required for full relaxation because of the uncertainty as to the exact point at which the curve of relaxation first touches the base line.

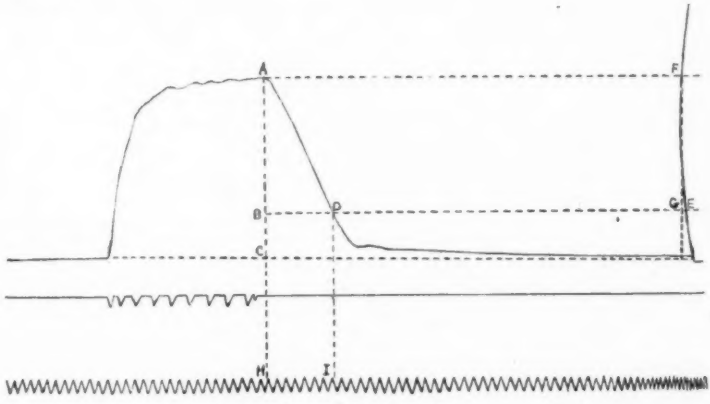

FIG. 1 is a tracing of the kymograph record of a typical contraction of the normal rat's gastrocnemius (left gastrocnemius of Tendon Rat 4). It illustrates the procedure followed in analysing the records.

The relaxation time was corrected for error due to the arc of the lever, as follows. The line AF, parallel to the base line, was drawn through A to the arc FE. A perpendicular to the base line was dropped from F. Where it

intersected the line BE, a point G was obtained. The distance GE represented the distortion due to the arc, and the time required for the kymograph to travel this distance measured at H, is the error due to the arc of the lever. The true time for three-fourths relaxation was then obtained by adding or subtracting the correction from the value previously found. Where, as was usually the case, this correction was less than one two-hundredth of a second it was disregarded in the calculations.

The relaxation rate was determined by dividing the distance AB in mm. by 5, the magnification of the lever, and by the corrected time HI in hundredths of a second. In order to determine the amount of shortening in the muscle during contraction the height of contraction AC was divided by the magnification of the lever.

RESULTS.

The muscle contractions were recorded with an isotonic rather than an isometric lever. It was thought that only by allowing the muscle full opportunity to shorten would the muscle, already shortened as a result of tenotomy or the action of tetanus toxin, betray its inability to undergo the normal amount of shortening when stimulated. The strength of the muscle, that is, the tension it could develop against an isometric lever, would be quite another matter. And, since, as Fulton[20] has pointed out, the phase of relaxation is a purely passive one, dependent on the viscosity of the muscle, the isotonic record, which allowed full shortening of the muscle and hence a large amount of elongation during relaxation, would be more likely to reveal by a slowing of the relaxation rate any change in viscosity of the muscle which might underlie the contracture.

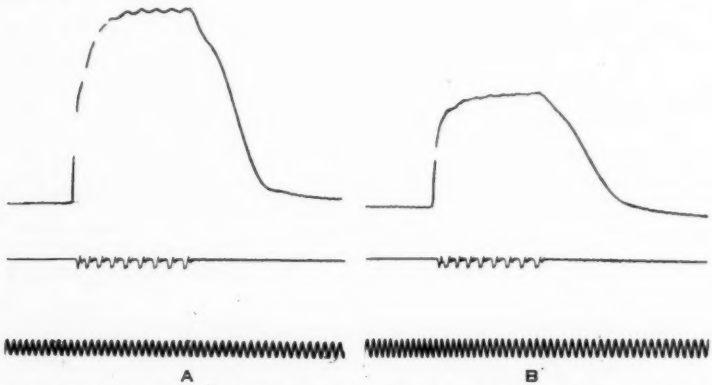

FIG. 2 is taken from the kymograph record of the experiment on a rat with tetanus contracture in the right hind leg (Rat T-5). The secondary coil was set at 9 and the time was marked in hundredths of a second. *A* is the tracing made by the normal left gastrocnemius, and *B* that made by the right gastrocnemius which was in myostatic contracture. The height of contraction and the rate of relaxation are less on the side of the contracture.

As is shown by Fig. 2, the height of contraction and the speed of relaxation are reduced in the muscle in tetanus contracture as compared with the uninjected muscle of the opposite side. The results of this series of experiments are summarized in Table 1. Several contractions were recorded from each muscle, analysed, and the data averaged to give the figures recorded in this Table. Analyses of individual contractions from one experiment are recorded in Table 2. The average amount of shortening of the gastrocnemius in tetanus contracture was 4·66 mm. as compared with 7·38 mm. in the control. In a series of six normal muscles the average shortening amounted to 7·52 mm. as shown in Table 3. It is not surprising that muscle in tetanus contracture failed to shorten during contraction as much as normal muscle since, as shown by Table 4, the fibres of the gastrocnemius on the side of the injection had become fixed in a shortened state and were only about one-half their normal length.

TABLE I.

Summary of data on the contractions of the gastrocnemius muscles in response to faradic stimulation of the sciatic nerve in animals with local tetanus.

	Injected Leg.			Control Leg.		
Animal Number.	Muscle shortening in mm.	Time for ¾ relaxation in 1/100 sec.	Rate of relaxation in mm. per 1/100 sec.	Muscle shortening in mm.	Time for ¾ relaxation in 1/100 sec.	Rate of relaxation in mm. per 1/100 sec.
Rat T2 ...	4·73	11·60	·305	6·50	12·00	·406
Rat T3 ...	2·66	5·00	·399	7·90	11·00	·538
Rat T4 ...	5·45	10·75	·380	7·40	11·75	·472
Rat T5 ...	4·88	9·00	·407	7·50	8·66	·649
Rat T6 ...	4·42	14·00	·236	7·53	8·00	·705
Rat T7 ...	5·87	10·14	·433	7·46	9·33	·599
Average...	4·66	10·08	·360	7·38	10·12	·561
Cat T1 ...	3·03	18·0	·126	4·85	12·85	·282
Cat T2 ...	1·52	12·2	·093	3·52	16·37	·161
Average...	2·27	15·1	·109	4·18	14·61	·221

There was not much change in the time required for three-fourths relaxation. This, when expressed in one-hundredths of a second, was 10·08 for the muscle in contracture, 10·12 for the control and 9·45 for the normal muscle (Tables 1 and 3). But due to the differences in height of contraction or, to put it otherwise, in the amount that the muscle had to elongate during relaxation, there was a marked difference in the rate of relaxation in spite of the

similarity of the times involved. The rate of relaxation in the injected leg was ·360 mm. per one-hundredth of a second, in the control leg ·561 and in the normal leg ·603. The decrease in the height of contraction and in the rate of relaxation is more marked when the comparison is made with the normal than when it is made with the control muscle of the opposite side because the tetanus toxin had some effect on the control muscle.

TABLE II.

Analysis of kymograph records of the contractions of the gastrocnemius muscle of the right (injected) hind leg of Rat T-4 in response to indirect and direct faradic stimulation.

Indirect Stimulation.				Direct Stimulation.			
Position of Secondary Coil.	Muscle shortening in mm.	Time for ¾ relaxation in 1/100 sec.	Rate of relaxation in mm. per 1/100 sec.	Position of Secondary Coil.	Muscle shortening in mm.	Time for ¾ relaxation in 1/100 sec.	Rate of relaxation in mm. per 1/100 sec.
9	5·6	12	·350	7	4·2	7	·450
9	5·6	12	·350	6	4·8	10	·360
9	5·4	10	·405	5	5·6	10	·420
9	5·2	9	·433	5	5·6	10	·420

The two cats were in a more advanced stage of tetanus. Cat T-1 had received 1½ cc. of tetanus toxin into one hind leg 7 days before the experiment and Cat T-2, 1 cc. 8 days before. The average muscle shortening during contraction on the side of the injection was 2·27 mm. as compared with 4·18mm. for the control muscles, and 6·10 mm. for a series of 3 normal muscles; and the average rate of relaxation was ·109 mm. per one-hundredth of a second as compared with ·221 for the control and ·254 for the normal (Tables 1 and 3). It will be noticed that throughout both tables the rate of relaxation was considerably greater for the rat than the cat. Although the circulation in the muscle was undisturbed in every case the cat muscle seemed to suffer more from exposure than the rat muscle did.

It is clear from these experiments that a muscle which is already shortened in tetanus contracture cannot undergo as much shortening in a nerve-muscle tetanus as can a normal muscle, and that its rate of relaxation is reduced. This slowing of the rate of relaxation would, according to Fulton's[20] interpretation of the curve of relaxation as an expression of the viscosity of the muscle, indicate that in tetanus contracture the viscosity of the muscle is increased.

When a large dose of tetanus toxin is injected directly into the quadriceps extensor muscle of the cat there often occurs within 24 hours a setting of the muscle in a condition of only partial contraction. The knee is partly flexed, cannot be moved voluntarily and offers considerable resistance against passive flexion to a more acute angle. This and other interesting manifestations of

TABLE III.

Analysis of kymograph records of the contractions of the gastrocnemii muscles after section of the right tendo achillis and the development of contracture.

	Right Leg, Tendon Cut.			Left Leg, Normal.		
Animal Number.	Muscle shortening in mm.	Time for ¾ relaxation in 1/100 sec.	Rate of relaxation in mm. per 1/100 sec.	Muscle shortening in mm.	Time for ¾ relaxation in 1/100 sec.	Rate of relaxation in mm. per 1/100 sec.
Rat Tend. 1	5·56	7·20	·579	7·76	9·20	·632
Rat Tend. 2	5·20	6·50	·600	7·33	8·16	·673
Rat Tend. 3	4·70	6·25	·564	7·43	10·50	·530
Rat Tend. 4	4·47	5·75	·582	7·05	8·25	·640
Rat Tend. 5	3·27	5·00	·490	7·00	11·00	·476
Rat Tend. 6	4·40	6·00	·550	8·60	9·62	·670
Average ...	4·60	6·11	·560	7·52	9·45	·603
Cat Tend. 1	3·80	11·50	·248	4·95	16·75	·221
Cat Tend. 2	5·10	21·00	·182	6·57	28·37	·173
Cat Tend. 3	6·45	10·85	·445	6·80	13·83	·368
Average ...	5·11	14·45	·291	6·10	19·65	·254

local tetanus will be discussed in another paper. Here we only wish to call attention to the fact that as the animal walks about there is a stiffness and apparent paresis of the affected limb. It must have been some such observations as these that led Fröhlich and Meyer[8] to state that in tetanus contracture central motor innervation is weakened or paralysed. The failure of the gastrocnemius affected with tetanus to shorten as much as the normal controls might be interpreted as evidence of such a paresis if it were not known that the muscle fibres were in a shortened state to begin with and could not therefore be expected to contract as much as they otherwise would.

In order to rule out the possibility that the defective contraction was due to an impairment of the nerve or nerve-endings, a comparison was made of the effect of direct and indirect stimulation in four experiments. In no case was there any evidence of involvement of the nerve or nerve-endings. The results obtained with Rat T-4 are given in Table 2. With the secondary coil at 9 the strength of the induction shocks was sufficient to produce a maximum contraction through the nerve. But on direct stimulation, because of the greater resistance in the circuit, the secondary coil had to be moved up to 5 in order to secure a maximum response. When a full response was obtained, the results of direct and indirect stimulation were similar both as

to the amount of shortening and the rate of relaxation. This demonstrates that the innervation was intact and shows that the alterations in function already noted were due to changes in the muscle fibres.

Within fifteen days after section of the tendo achillis the rat gastrocnemius goes into a contracture very similar to that caused by tetanus toxin. As shown in Table 3, the affected muscles shortened on the average 4·6 mm. during contraction as compared with 7·52 mm. for the normal leg. The time required for three-fourths relaxation was distinctly less on the side of the tenotomy, but because of the greater height of contraction of the normal side the rate of relaxation figures out to be about the same on the two sides. The difference between ·560 and ·603 in favour of the normal muscle is probably not significant. In the contracture following tenotomy, while the ability to undergo further shortening during contraction is reduced to the same extent that it is in tetanus contracture, the rate of relaxation is practically normal. There is therefore no evidence that in this sort of contracture there is any increase in the viscosity of the muscle. So far as one can judge from the character of the recorded contractions the muscle in contracture following tenotomy has merely taken on a new and shorter resting length.

In Table IV there are given the data relative to the length and weight of the rat's gastrocnemius and the length of a selected, easily identifiable group of fibres in that muscle. In the first series the right gastrocnemius was in contracture due to tendon section. In the second series the right gastrocnemius was in contracture due to tetanus. The left side served as control in both series; but, because the action of the tetanus toxin is not strictly confined to the side of the injection the figures for the control leg in the tendon series are to be regarded as representing the normal; and there was some shortening of the fibres in the control gastrocnemius of the tetanus series.

The measurements were made after the muscles had been fixed for 24 hours or longer in 10 per cent. formalin under a tension of 25 grams. The length of the muscle was measured from the attachment of the medial head on the femur to the last insertion of muscle fibres into the medial side of the tendon. The tendon was cut away at this level and the heads of origin separated from the femur. The weight of the muscles when thus isolated is recorded in the Tables. The length of the fibres was determined as follows. On the anterior surface of the outer head two longitudinal septa are visible separating three groups of muscle fibres. The middle group of fibres which runs obliquely between the two septa was put on a stretch by spreading the muscle out and pinning it down on a cork plate. The length of the fibres was then determined with dividers and a millimetre scale. The measurements were in reality not of individual muscle fibres but of small bundles of fibres. But histological examination showed that in these bundles the fibres ran without interruption through the entire length of the bundle.

TABLE IV.

Showing the effect of tenotomy and of tetanus contracture on the weight and length of the rat's gastrocnemius and on the length of the muscle fibres. Tend. designates the rats in which tenotomy was performed and T. those with tetanus. The left leg severed as control.

	Right Gastrocnemius, Contracture.			Left Gastrocnemius, Control.		
Rat.	Length in mm.	Weight in gms.	Fibre length in mm.	Length in mm.	Weight in gms.	Fibre length in mm.
Tend. 1 ...	27·5	1·480	6·5	—	1·870	11·8
Tend. 3 ...	24·1	0·820	6·1	27·5	1·175	9·5
Tend. 4 ...	25·5	0·831	4·1	29·2	0·935	10·1
Tend. 5 ...	26·9	1·180	3·5	31·8	1·535	9·0
Tend. 6 ...	26·0	1·535	4·9	30·2	1·730	—
Tend. 7 ...	22·0	0·820	4·9	25·9	0·943	9·1
Average...	25·3	1·111	5·0	28·9	1·364	9·9
T–2 ...	24·7	1·075	4·5	30·1	1·364	8·1
T–3 ...	28·2	1·494	4·8	31·0	1·494	9·0
T–4 ...	24·2	1·479	4·2	26·8	1·405	7·8
T–5 ...	25·0	1·210	6·8	28·5	1·242	10·1
T–6 ...	24·7	1·304	6·0	28·6	1·390	10·0
T–7 ...	28·7	1·470	6·6	29·5	1·569	7·8
Average...	25·9	1·338	5·48	29·08	1·410	8·8

It will be seen from Table 4 that after tendon section or the administration of tetanus toxin the length of the muscle in contracture is somewhat less than that of the control, but the difference amounts to only about 10 or 12 per cent. of the length of the normal muscle. The difference in the length of the fibres is more striking, amounting to 45 or 50 per cent. of the length of the same fibres in normal muscle. The muscle in the control leg in the tenotomy series is taken as the normal standard, since the tetanus toxin circulating in the blood stream caused a slight shortening of the fibres in the control leg of the tetanus series. The obliquity of the fibres in the gastrocnemius accounts for the much greater percentage of shortening in the fibres than in the muscle as a whole.

The muscles in tetanus contracture have a normal weight. In the six experiments the average weight of the gastrocnemius on the side of the injection amounted to 1·338 grams as against 1·410 grams on the control side and 1·364 on the normal side of the tenotomy series. These differences are much less than the differences in the individual muscles of a series and are therefore of

no significance. In tetanus contracture, therefore, the weight of the muscle is not affected either by atrophy or swelling; on the contrary, the weight remains normal.

After tendon section, however, an appreciable amount of atrophy occurs. As a result of tenotomy the weight of the right gastrocnemius was reduced to an average value of 1·111 grams as compared with the average of 1·364 grams for the corresponding muscles of the opposite side in the same animals.

Lipschütz and Audova[21] studied the atrophy resulting from tenotomy in rabbits, found that it was nearly as great as that caused by nerve section, and considered that the atrophy was due to inactivity. After tenotomy as after nerve section they found the relative amount of water in the atrophied muscle distinctly greater than normal.

It is, however, questionable if inactivity forms an adequate explanation of the atrophy following tenotomy. Meyer[22] has shown that if a flexed joint is put up in a plaster cast the flexor muscles atrophy but the extensor muscles hypertrophy. Froboese[23], who studied these hypertrophied muscles for Meyer, found that except for the hypertrophy of the individual muscle fibres they presented a normal appearance under the microscope. These results seem to show that moderate tension causes hypertrophy and relaxation causes atrophy of muscles in an immobilized limb.

The atrophy of a muscle following its release from tension after tenotomy and the setting of the same muscle in a condition of permanent shortening are phenomena which are closely related. But neither the atrophy nor the setting has as yet been satisfactorily accounted for. Myostatic contractures form a field in which further investigative work is needed both because of the practical importance of the subject and because a solution of the problems there presented would throw light on the function of normal muscle.

SUMMARY.

Contractures may be separated into two groups depending on whether or not they are maintained by a continuous discharge of nerve impulses into the muscle. Hypertonic contractures, including those developing in the course of spastic paralyses and those of reflex origin, are dependent on a continuous tonic innervation of the affected muscles. On the other hand, myostatic contractures, including those which follow tenotomy or the immobilization of a limb in a plaster cast, are due to a shortened condition of the resting muscle and are entirely independent of the nervous system. Very little is known about the nature and cause of the changes in muscle which are responsible for its becoming set in a shorter than normal resting length. The work of Meyer, Fröhlich and Meyer, and Spiegel and Shiboya, serves to define the problem, but contributes little to its solution.

A muscle in myostatic contracture due to tenotomy or to the action of tetanus toxin maintains its shortened state after all nerve impulses have been cut off by the section of the motor nerve or by the death of the animal. It

has acquired a new and shorter than normal resting length. Moreover, its extensibility has been decreased and this affects only its ductility or "Nachdehnung" while its elasticity remains normal.

Microscopic sections of such a shortened muscle reveal no structural changes which could be responsible for keeping it shortened. There is no increase in connective tissue and little or no change in the appearance of the muscle fibres. After tetanus contracture has persisted for several weeks, secondary changes, such as an increase in the number of nuclei, occur, but these appear late and are in no way responsible for the shortening.

In tetanus contracture and in that caused by tenotomy a selected group of fibres in the rat's gastrocnemius shortened 45 or 50 per cent. of their normal length; but owing to the obliquity of the fibres the muscle as a whole underwent a shortening of only 10 or 12 per cent. The weight of the muscle remained normal in tetanus contracture, but after tenotomy the average weight was reduced to 1·111 grams from an average normal of 1·364 grams.

Kymograph records show that a muscle in myostatic contracture cannot shorten as much as a normal muscle during nerve-muscle tetanus. In these experiments the average normal shortening of the rat's gastrocnemius during contraction was found to be 7·5 mm. and this was reduced to an average of 4·6 mm. for the muscles in contracture. While the capacity for shortening during nerve-muscle tetanus was decreased to the same extent in both types of experimental myostatic contracture, the rate of relaxation was unaffected by tenotomy but was considerably reduced by tetanus toxin.

The height of contraction and the rate of relaxation of a muscle in tetanus contracture are the same whether the muscle is stimulated directly or through its nerve. This shows that the defective contraction cannot be explained on the basis of an involvement of the nerve or nerve-endings.

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LOCALISED CRANIAL HYPEROSTOSIS IN THE INSANE.

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Of the various anomalies of texture which may be found in the cranial bones of the insane, generalised thickening and sclerosis is perhaps the commonest, and is seen in its most typical form in the dementias of epilepsy and chronic insanity. On the other hand, thickening of the cranial vault, limited to one particular area or bone, and accompanied in some cases by the formation of osteophytes, appears to be a rather rare condition. Usually it is the frontal bone which is affected in this way, and its precise appearance is subject to considerable variation. The construction of the calvaria may be fairly normal, the diploë being represented, but the two tables much thicker than normal. In other cases the increase is confined to the inner aspect, which has either a craggy embossed, or smooth surface. In a third type the main part of the enlargement consists of diploë limited by well defined tables. The amount of thickening is often considerable, and Shattock[1] mentions a case in which the maximum thickness of the frontal bone was 1·9 cm. According to Lawford Knaggs[2] in some cases the condition appears to be clearly inflammatory, the inner surface presenting a rugged, raised, irregular appearance suggestive of periosteal deposit, with channels directed towards the superior longitudinal sinus that evidently mark the position of dilated veins.

One of the earliest descriptions of localised hypertrophy is contained in Bevan Lewis' text book of mental diseases[3], in which reference is made to fifty-four cases: in thirty-one of these the frontal was the site of the bony increase; in seven cases the occipital, and in six cases the parietal were the regions affected. Clouston[4] has also recorded a particularly good example of hypertrophy limited to the frontal bone. The patient had suffered from alternating insanity of over twenty years' duration and the skull cap showed "enormous deposits of new osseous tissue distributed in an irregularly modulated way on the inner table of the skull." Concerning the origin of the condition Clouston writes: "This seems to be an aggravated example and type of what is almost universal in chronic insanity with periods of excitement. It is a proof of the structural effects of such repeated congestions of the branches of the carotid artery, even in the hardest tissue, and may be fairly considered to be of the same nature as the brain changes in the same cases, which are not so evident, but are no doubt far more important. The atrophy of the anterior lobes of the brain that usually accompanies such bony thickenings and deposits probably helps their growth, they being thus compensatory to some extent, like the increased cerebrospinal fluid."

Beadles[5], in an interesting paper devoted to the morbid anatomy of the cranium, remarks that bony deposits when distinctly limited are usually found on the frontal bone. The condition, he states, is usually seen in cases of chronic mania or such as have passed into dementia, and frequently these patients have been the subjects of marked periodical excitement or alternating insanity. He agrees with Clouston that the condition is brought about by repeated temporary attacks of congestion of the brain and its coverings. A rather more adequate discussion of the pathology of frontal hypertrophy is given by Shattock, who describes in detail the appearances of a number of museum specimens. Among these there may be noted:—

1. The calvaria of an epileptic imbecile with a thickness of 1·9 cm. at the anterior part of the frontal bone.

2. A specimen with marked thickening of the inner tables of the frontal bone from "a stoutly built woman," age 37, who had epileptic fits every three or four days, and whose dementia was accompanied by attacks of violence.

3. A case of recurrent mania of fifteen years' duration, the patient dying at the age of 61. An irregular bony deposit was found on the inner portion of the frontal bone to the extent of three-quarters of an inch.

4. A female, insane for thirty-two years, who died in her 77th year. She is said to have been very stout. The calvaria was extremely thick, with little diploë; irregular projecting bony deposits were present on the inner surface of the frontal bone.

5. A female, age 55, with a history of alcoholism. Death occurred from dementia following mania of nine months' duration. The calvaria was thickened and congested, and the internal surface of the frontal bone very irregular from the presence of new bony growth.

6. A female, age 58, with a history of dementia following recurrent mania of over twenty years' duration. The calvaria was of average thickness, except at its frontal portion where the inner surface had a bossy or craggy surface.

7. A female dying at the age of 67 from senile dementia. Calvaria dense and heavy, with pronounced thickening on the inner aspect of the frontal bone.

8. A female, age 52. Chronic mania of seven years' duration. The calvaria showed slight general thickening, and a more pronounced hypertrophy in the frontal region with a craggy internal surface.

9. A female, age 72, who suffered from senile dementia. Thickening was present in the frontal region, which had a slightly irregular inner aspect.

10. Two specimens from Guy's Hospital Museum. One, from a female, age 55, with a long history of insanity, and the other, from a female, age 48, whose insanity was of eight years' duration. The brain was particularly small, and "marked with depressions corresponding with the elevations upon the inner aspect of the frontal region."

In the cases referred to above, little information is conveyed concerning either the physical state of the patients during life or the post-mortem findings, and it will therefore be of some interest to give a more detailed description of several cases I have had the opportunity of seeing during the course of the last fifteen years.

PERSONAL SERIES.

CASE 1. J. F., female, age 48, was admitted to an asylum on November 2, 1899, suffering from melancholia. There was a history of three previous attacks, the first of which had occurred at the age of twenty-one. No physical or neurological signs of importance were noted on admission, but her photograph showed a commencing lobulation of the skin on either side of the nose. At first she was fretful and querulous, and expressed a delusion that she was persecuted by a man. Her mental disorder ran an unfavourable course, and very soon she became dull, listless and apathetic, showing considerable impairment of memory. After two years a state of mild dementia supervened, and she was then reported to have become very ponderous and slow in her movements, and to have gained enormously in weight.

In November, 1911, I made the following notes on her condition: She is an extremely fat woman, weight 18 st. 4 lbs.; height 5 ft. 10 in. The distribution of adipose tissue is general, but the panniculus is particularly abundant on the abdomen, and there is a tendency to lobulation on the thighs. Her face has a swollen appearance, as striking as that of myxœdema, with large fatty swellings on its surface, giving rise to a marked narrowing of the palpebral apertures; there is no pitting on pressure. At first glance her bloated œdematous appearance suggests the presence of an intrathoracic neoplasm, but there is no cyanosis, her skin being pale in colour. Patches of yellowish pigmentation are present on the face and limbs, and in the former situation there are numerous small tags of loose skin, especially marked on the eyelids. The hair is scanty, harsh and dry, and is turning grey; on the upper lip and chin there are numerous coarse hairs, many of them being more than one inch in length; pubic hair normal in distribution and amount. No crescents are visible on her nails. There are no skeletal changes. Her voice is loud, harsh, and guttural. Appetite not excessive. Pulse 90, and of low tension. Heart sounds somewhat muffled; blood examination shows no abnormality. Lungs healthy. Temperature subnormal. Urine: sp. gr. 1010, pale in colour, no abnormal constituents; marked polyuria. Pupils equal, contracted, and reacting normally to light and on convergence. Special senses, cranial nerves, sensation and motor power normal. Reflexes normal.

Mental State.—Her general intelligence is difficult to estimate as she is suspicious and hostile in manner. When spoken to she uses obscene language, and pays no attention to questions. Although very torpid and sluggish she is able to do useful work of a simple nature, such as mending articles of clothing.

Subsequent History.—A noticeable feature during the last months of the patient's life was a marked polyuria. She passed large quantities of pale urine, free from albumen or casts. Owing to her incontinence the daily excretion could not be estimated. On November 6, 1913, she developed acute bronchitis, and on the following day her condition was one of extreme dyspnœa. At 9.30 p.m. she became unconscious and died on November 8, at 6.20 a.m. The left eye showed chemosis some hours previous to death.

Post-Mortem Appearances.—Necropsy was held five hours after death. The abdominal wall was several inches thick owing to the great deposit of fat, which was normal in colour. The calvaria was thickened, and exceedingly dense. The inner table was covered with large, irregular bony protuberances, most numerous on the frontal bone (Fig. 1), but also

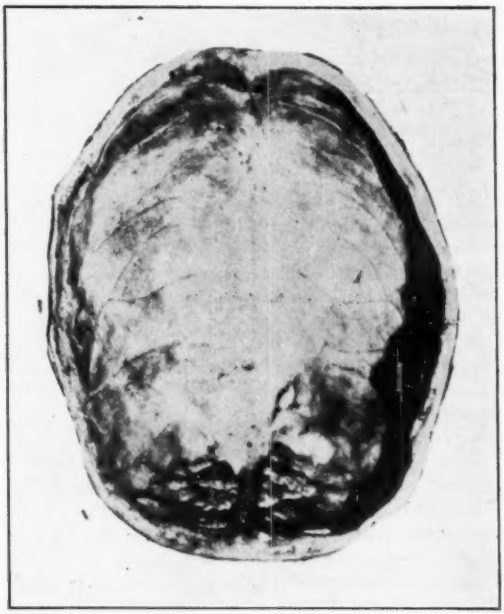

Fig. 1. Case I. Showing bony masses on the inner table of the frontal bone.

present on parts of the parietals and occipital bone. The diploë was reduced in amount. The dura mater, which was slightly thickened and adherent to the inner surface of the skull, contained a large bony plate, nearly a quarter of an inch thick and more than one inch long, in the anterior end of the falx cerebri. Pia mater congested. The brain weighed 1,150 grm. and showed no naked-eye abnormality; the cerebral sinuses and veins were normal. The pituitary body was unusually firm and slightly enlarged. The heart showed an extreme degree of fatty infiltration, and the lungs evidence of acute bronchitis and œdema. The gallbladder was filled with cholesterin stones. The ovaries appeared shrunken and cirrhotic; the suprarenals soft and friable. The kidneys and other organs showed no obvious departure from the normal.

Microscopic Examination of the Pituitary.—For the histological examination I am indebted to Professor T. P. Herring, who kindly sent me the following report:

"*Anterior Lobe*: There is marked sclerosis, a condition, in fact, resembling cirrhosis of the liver; there is marked deficiency in the chromophil elements and a strangulation of the cells by connective tissue.

"*Pars Intermedia*: The arrangement is rather unusual, and there is also a considerable spread of its cells into the pars nervosa. There is also a good deal of lymphoid tissue in it.

"*Pars Nervosa*: No abnormality save invasion by the pars intermedia."

The right half of Fig. 2 illustrates the microscopic appearance of the anterior lobe; the left half shows for comparison the anterior lobe of a normal or nearly normal pituitary.

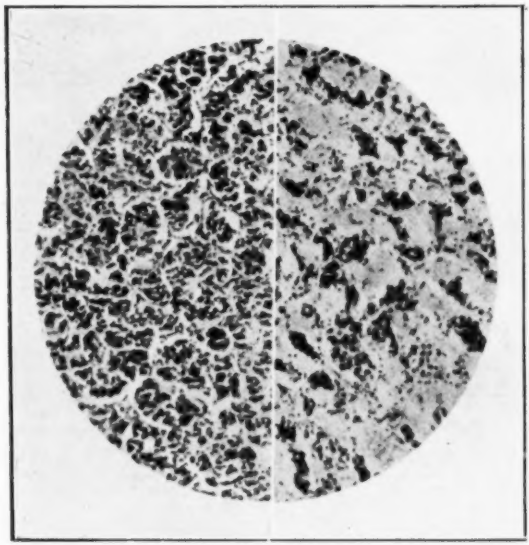

Fig 2. Right half shows sclerosis of anterior lobe of pituitary from Case I. Left half shows for comparison the cells of a normal anterior lobe. Both × 90.

Case 2. A. B., female, age 47, was admitted to hospital on May 17, 1911, suffering from "insanity with gross lesions" of six years' duration. She was extremely stout, her weight being 15 st. 3½ lbs. Height 5 ft. 1½ in. The notes on her physical condition are scanty. The presence of heart disease, and deformity of the fourth and fifth digits of the right hand are recorded, and reference is made to a stroke, said to have occurred seven years previously. Mentally she was in a state of dementia, being disorientated in time and place, faulty in her habits, and unable to occupy herself. In the ensuing fifteen years no change in her mental condition took place. Syncopal attacks occurred at infrequent intervals, and in 1921 the presence of right hemiplegia is recorded. This seems to be an error, there being no evidence of paralysis a year later. Her weight varied between twelve and fifteen stones.

On December 7, 1927, she became suddenly ill with distressed breathing, feeble rapid pulse, and pyrexia. Jaundice was present next day, and she died at 11.25 a.m. on December 9.

The autopsy was held on the morning of December 12, and owing to the cold weather the body was in a fairly good state of preservation.

The body was that of a fat female, with a heavy moustache and numerous coarse hairs on the chin. The distribution of fat was most marked on the lower part of the abdomen and thighs; girth at umbilical level 39½ in. The skin had the greenish hue of jaundice. No deformity of the limbs was noted. The head measured 22½ in. in circumference. The calvaria was thicker than normal, and this thickening was especially pronounced in the frontal region, being apparently due to overgrowth of cancellous tissue. Over a rather limited area of the frontal bone were irregular masses of dense bone. The sella turcica (Fig. 3) was much enlarged, chiefly at the expense of the posterior clinoid

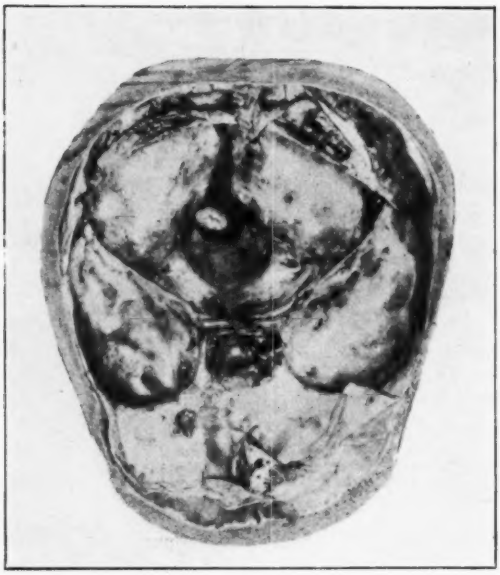

Fig 3. Base of skull from Case II. showing enlargement of sella turcica.

processes, which together formed a thin plate which fractured easily. The pituitary gland was enlarged and fitted tightly in its fossa; its lateral aspects were compressed and flat. It was firmer than normal and weighed 0·875 grm.

The brain weighed 44 oz. Convolution pattern fairly complex; considerable wasting of the frontal and parietal convolutions, particularly in the left hemisphere. The right cerebellar hemisphere was markedly smaller than the left, and was associated with slight atrophy of the right olivary body. The arteries at the base showed a moderate degree of atheroma.

The lungs exhibited old pleural adhesions and scarring at the right apex; the heart weighed 6½ oz., and showed fatty degeneration, and disease of the cusps of the mitral valve. The abdominal wall was heavily loaded with fat; the peritoneal cavity contained about 12 oz. of purulent material. A fistula existed between the apex of the gallbladder and the hepatic flexure of the colon. Facetted stones were present in the thickened gall-bladder and in the common and cystic ducts. The liver weighed 62 oz., being moderately enlarged. On section the bile ducts appeared dilated and contained pus. The spleen and kidneys showed no noteworthy change. The ovaries could not be identified. Cause of death: general peritonitis following cholecystitis and cholangitis; fatty degeneration of the heart.

Microscopical Examination of the Pituitary.—The gland capsule is thickened, and lying on its external surface at the posterior pole of the organ is a collection of epithelium-like cells resembling those of the pars intermedia.

Anterior Lobe.—This appears considerably enlarged and shows a very marked increase in the amount of reticular connective tissue between the cells (Fig. 4). The latter

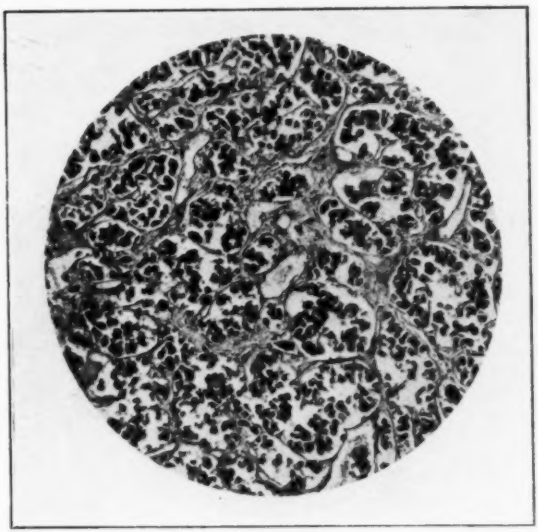

Fig. 4. Case II. Anterior lobe of pituitary, showing atrophy of cells and increase of connective tissue. × 90.

are for the most part shrunken and reduced in number, there being a noticeable loss of the arrangement of cells in compact masses such as is seen in the normal gland. Granular oxyphil cells are fairly numerous, especially at the posterior end of the lobe.

Pars Intermedia.—There is less connective tissue in this area and in one situation the cells have spread into the pars nervosa.

Pars Nervosa.—This lobe is also somewhat enlarged, and the neuroglia more fibrous than normal. The interglandular cleft shows masses of colloid material.

Case 3. C. K., widow, age 62, was admitted with a diagnosis of recurrent melancholia on June 2, 1909. Four years later she was noted to be childish, voluble and unable to remember recent events. A note made in February 1915 described her as more demented and incoherent. On December 9, 1919, she was noted to be walking with difficulty, and was unable to reply to questions or to obey commands. Slight paresis of the left arm and leg was present, and on January 1, 1920, her illness terminated with symptoms of uræmia.

A post-mortem examination was held eleven hours after death. The body was that of a very fat old woman, 11 st. 12 lbs. in weight. Height 61 inches. A considerable growth of hair was present on the upper lip and chin; the texture of the skin was smooth. The calvaria was dense and thickened, and on the inner table of the frontal bone there were dense nodular masses of bone. The brain weighed 1,160 grm. and apart from slight atrophy of the frontal gyri showed no abnormality. The heart and liver were fatty, the kidneys granular and contracted, and the lungs in a condition of hypostatic pneumonia. The pituitary gland appeared slightly enlarged; weight 0·56 grm.

Microscopic Examination. The microscopic appearances bore a close resemblance to those noted in Cases 1 and 2. The enlargement affected principally the anterior lobe; its cells were atrophied and surrounded by numerous strands of connective tissue (Fig. 5). The pars intermedia and pars nervosa showed no abnormality.

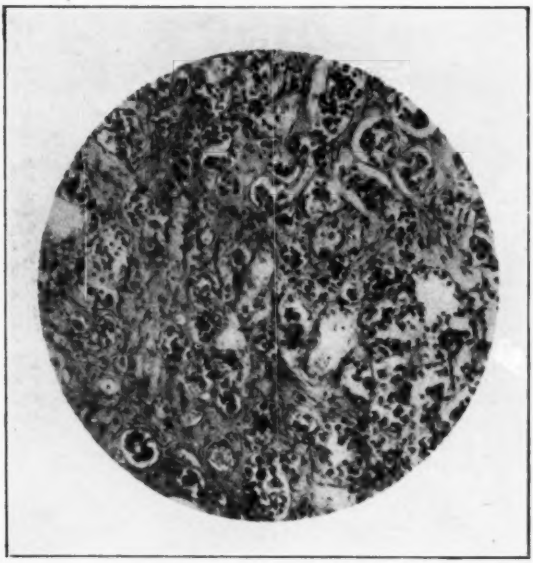

Fig. 5. Case III. Anterior lobe of pituitary showing appearance very similar to those in Case II.

In addition to the above personally observed cases I have had the opportunity of examining a number of museum specimens of frontal hypertrophy, and am able to give the following details of two examples. The clinical notes on the first case, the date of entrance into the asylum being 1875, were very meagre. The second was more adequately described.

Case 4. F. S., male, age 19, was admitted on May 25, 1875, suffering from epileptic insanity. He was described as "A short heavy-looking lad of good fatness." Mentally he was childish and peevish. His fits averaged two per week, and occurred only in the day time. He was employed in outdoor labour, until he finally became dangerous and partially demented. On July 9, 1902, he died in status epilepticus. At the post-mortem examination a tuberculous cavity was found in the right lung. The brain weighed 52 oz. and showed atrophy of the convolutions in the motor and frontal regions. Diffuse milkiness of the pia-arachnoid was also noted. The skull cap was exceedingly thick and dense, showing large deposits of bony tissue on the inner table of the frontal bone. These were distributed in an irregular fashion and were so hard that the chisel made little impression on them.

The cancellous tissue is principally responsible for the great thickness of the frontal bone (Fig. 6).

Case 5. D. P., mechanic, age 51, was admitted to the asylum on August 3, 1885, suffering from melancholia; he had attempted to cut his throat. He was discharged after some months, but was soon re-admitted with a return of his suicidal tendencies and symptoms of melancholia, which became chronic. In 1892 he was described as having become very fat, and a further note made ten months later stated that he complained a great deal of frontal headaches and was now much stouter. Finally, he succumbed to capillary bronchitis on October 31, 1894.

Fig. 6. Skull cap from Case IV. Note hypertrophy of frontal bone; in this case the diploë is enlarged.

At the autopsy it was noted that he was extremely corpulent, measuring 53 inches round the abdomen; his height was 5 ft. 3 in. The heart weighed 18 oz. and was loaded with fat; the base of the aorta was atheromatous, and the aortic valve incompetent. The mucous membrane of the bronchi and bronchioles was acutely inflamed and the bronchial glands enlarged. The liver weighed 68½ oz. and was pale and fatty. The other organs showed no noteworthy change. The brain weighed 52½ oz. Slight dilatation of the ventricles was present. The pituitary body was not examined. The skull cap was extremely dense and thick, with irregular nodulated masses on the inner surface of the frontal bone (Fig. 7).

Fig 7. Case V. Cranial vault showing bony masses on inner table of frontal bone.

DISCUSSION.

As already noted, in the earlier papers devoted to this subject considerable importance is attached to the form of insanity displayed by patients who show hypertrophy of the frontal bone, and Beadles claims that the majority of, if not all, the cases occur in patients who have suffered from chronic mania or the dementia following mania. Analysis of the cases quoted above shows, however, that this is by no means invariably the rule, and other mental states such as melancholia or epilepsy may be present. For example, in case 1 of the author's series there was nothing in the history to suggest the "repeated attacks of congestion of the brain" said to exist in alternating insanity. Indeed, if the diagnosis can be trusted, in the early stages of her illness the patient suffered from recurrent melancholia, which according to Beadles is usually associated with thinning of the cranial vault. Even if the cerebral congestion accompanying mania occurred in every case, it would be difficult to explain how this could influence the supply of blood to the frontal bone, for the arterial supply of the calvaria is distinct from that of the brain, so that an active hyperæmia of the one does not necessarily involve that of the other. According to Shattock, the only way in which cerebral congestion could produce congestion of the calvaria would be by the engorgement of the cerebral veins which open into the various sinuses. It is, he thinks, conceivable that in this way a secondary congestion of the bones might, after the manner of Bier's method, bring about an overgrowth of their tissue.

A second factor supposed to be concerned in the production of localised calvarial thickening is recession of the subjacent cerebral substance accompanying the dementia seen in some of these cases, but how exactly atrophy or shrinkage of the brain can stimulate the formation of new bone is not easy to discern.

Atrophy of the cerebral convolutions is by no means uncommon in the chronic insane, but hypertrophy limited to the frontal bone is decidedly rare. Moreover, during the period of childhood or youth a cessation in the growth of any portion of the brain is not followed by thickening of the corresponding area of the skull. Again, shrinkage of the brain in adults is readily compensated for by an increased formation of cerebrospinal fluid, and the old assumption of a negative pressure within the cranium in cases of brain atrophy is hardly tenable to-day.

The connection between toxins circulating in the blood and the enlargement of certain bones, such as is seen in hypertrophic pulmonary arthropathy, has given rise to the suggestion that the condition under discussion may be toxic in origin. In certain cases a history of alcoholism is obtainable, and Shattock suggests that alcohol may serve as a second though subsidiary factor in producing congestion of the calvaria, or may act in the less direct manner of bringing a portal infection and a slow process of toxæmia, the congestion of a part with toxic blood being possibly more productive of overgrowth than one with normal. The principal objection to this view is the infrequency

of a history of toxæmia or alcoholism, and the occasional presence of frontal hypertrophy in mental defectives who have had no opportunity of acquiring the alcohol habit.

In the cases reported above stress has been laid on the presence of marked obesity. Possibly in some this can be attributed to the mental hospital regime, but in at least three the adiposity was of the pituitary type, being subsequently found to be associated with definite pathological changes in the pituitary body of an unusual type, and consequently the question arises whether the pathological and clinical features were causally related. The solution of this problem is by no means easy, for although nearly all observers are agreed that disease of the pituitary or of the neighbouring nervous structures at the base of the brain can effect profound alterations in the direction of overgrowth or undergrowth, the task of correlating the different types with pathological lesions is one of extreme complexity. In acromegaly, the prototype of hyperpituitarism, thickening of the calvarial bones is not a usual feature, although it is interesting to note that in the case of the acromegalic giant Charles O'Brien, whose skeleton is preserved in the museum of the Royal College of Surgeons, the skull shows marked thickening of the frontal bone. Nor does this condition appear to have been recorded either in Fröhlich's dystrophia adiposo-genitalis or in any of the other less well defined syndromes of hypopituitarism or dyspituitarism. The influence of the pituitary on the growth or rather the retarded growth of the cranial bones is, however, exemplified by the interesting syndrome of pituitary dysostosis, in which dwarfism, diabetes insipidus, and exophthalmos are associated with defective formation of certain bones of the skull. The condition occurs chiefly in young children, and is stated by Schüeller[6] to be probably an unusual type of hypopituitarism.

If hypertrophy of the frontal bone could be shown to be a constant, or even frequent, feature in acromegaly it would be feasible to explain its presence in the insane on the assumption that overstimulation, resulting, among other things, in the formation of new bone, gave place to symptoms of exhaustion before sufficient time had elapsed for other bones to become involved, the symptoms of exhaustion manifesting themselves by the development of marked obesity. This does not, however, appear to be the case, frontal hyperostosis in acromegaly being the exception rather than the rule. None the less, the association of this condition with marked obesity in the insane—and especially those of the female sex—seems to be too frequent to be merely accidental, and when considered in the light of the pathological changes in the three cases recorded above, gives grounds for believing that we are here dealing with a hitherto undifferentiated type of dyspituitarism.

REFERENCES.

[1] Shattock, *Internat. Med. Congress*, 1913, xviii (iii—Path. and Path. Anat.), pt. 2, 26.

[2] Lawford Knaggs, *The Inflammatory and Toxic Diseases of Bone*, Bristol, 1926.

[3] Bevan Lewis, *Text Book of Mental Diseases*, London, 1889.

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INHIBITORY EPILEPSY.

By S. A. KINNIER WILSON, London.

The fact that many of the phenomena of what is ordinarily understood as epilepsy are inhibitory and not excitomotor in character has scarcely received the attention it deserves. Yet the clinical neurologist who gives some consideration to the question cannot fail to be impressed with the comparative frequency, in epileptic cases, of symptoms not to be explained physiologically except by some process of inhibition. Loss of consciousness—what else is it than arrest of function, indeterminate though its mechanism may still to some extent be? Perhaps the most typical feature of the average case of petit mal is cessation of function of one or other form. The patient stops what he is doing, stares, drops what is in the hand, ceases speaking, falls, sits still and motionless, ignores questions or does not hear them, and so on—whatever the precise formula in a given case the major portion of the syndrome consists patently in the expression of some variety of inhibition. Similarly, in many severe fits the initial stages illustrate the action of the same physiological mechanism, and so do certain phenomena to be noted occasionally at a later period. For example, loss of sight—darkness, blindness—is not infrequent as an aura; loss of hearing, though less common, occurs similarly now and then; loss of speech, too, may develop at the stage of prelude. Initial muscular relaxation also deserves mention. Not a few post-convulsive symptoms usually attributed to exhaustion are better regarded as exemplifying inhibition.

Of no less interest is the question of stimuli of one or other sensory kind being succeeded not by motor excitation but by motor inhibition. Gowers is one of the very few writers who have alluded to this possibility. He was of the opinion that weakness associated with epileptoid seizures may in minor attacks be due to inhibition of the same centres as are discharged in severer attacks, and mentioned a case of Jacksonian epilepsy (typical, right-sided) in which minor fits consisted of inability to speak or move the right arm or leg, there being no convulsion. More significant, for the purpose of this communication, are two other cases referred to briefly by the same author. The first is that of a woman of 40, probably the subject of organic disease, whose attacks are thus described.

> "They begin with 'ticking in the right ear,' and this is followed by a painful sensation 'like hot needles running into the skin,' which passes down the side to the leg and foot and, after reaching the toes, returns up the leg and side and is felt in the arm, hand, and tongue. She mumbles and cannot speak for half an hour. There is no motor spasm, but as

soon as the sensation is felt in the leg and arm the side becomes extremely weak, so that, although ordinarily able to walk well, she becomes unable to stand and scarcely able to raise the arm."

The second case, reported with greater brevity, is that of a patient of 48: the attacks consisted in "pins and needles" in the left thumb, which passed up the arm to the shoulder and left scapula, with a sense of contraction in the arm. This became powerless, so that she dropped anything that was in the hand.

These and analogous cases may be taken to illustrate restraint or inhibition of motor centres by epileptic sensory discharges, a phenomenon which, as will be shown later, can be substantiated by other data well known to the physiologist.

A remarkable example of the same epileptic syndrome was under my close observation for a number of weeks, and is here narrated in detail.

PERSONAL CASE.

W.C., male, age 24, unmarried. Occupation, barman.

Family History: Mother, who died at age of 39, from heart disease, suffered from "fainting fits." No record of epilepsy or insanity in near or distant relatives.

Previous history: Always has had good health. Denies venereal infection. Is abstemious in regard to alcohol and has never been the worse of liquor. No rheumatism or rheumatic fever.

History of present illness: Six months ago, without any apparent cause, the patient began to suffer from curious attacks of tingling in the left hand and arm, and left side of the body, which have continued steadily and are now becoming more frequent. At first these occurred perhaps one or two in a fortnight, but now every two or three days. They may come at any time, but as far as he knows not during the night. In the attacks he never loses consciousness but he feels queer and "regular knocked."

Aura: Of this a very definite description is given, and it has always been the same. It begins by his eyes becoming "misty," not one eye more than the other, or before the other. He can see his way about but he cannot distinguish the forms of objects. He has noticed that the mistiness is chiefly on looking downwards; on looking to either side he sees constant "bright flashing stars" in the outer parts of the field. They are not coloured.

This mistiness of vision and these subjective visual phenomena continue for about 15 to 20 minutes before the attack proper commences.

Character: The attack commences by a sensation of tingling, of formication, of "wires" in the left fingers and hand. This is variously described by patient—it is as if "he had his hand in a stocking"; as if "it were entangled in string." In the earliest attacks the sensation did not pass the wrist, and patient was able to abate it by putting his hand into water, rubbing it, etc. But all the other attacks have been wider in range. The sensation passes up the left arm in a few seconds, then down the left side of trunk rapidly into leg and foot and toes; it then appears (in the more recent attacks) in the left side of the face (lower division). It plays along the lower jaw on the left side, and makes the left half of the tongue tingle.

The duration of the sensation varies from only a few seconds to some minutes. When it is over, it leaves arm, leg and face quite numb and "dead."

At the same time as the numbness appears patient loses all power in his arm and leg. Anything he has in his hand he will let drop; he will be unable to pick anything up with

the fingers. In a second or two the loss of power in the left arm and hand is absolute and he is quite unable to move it. Similarly with the leg, he is completely unable to move it at any joint.

The duration of this combined numbness and loss of power may be merely a minute or two, or as long as 20 minutes.

When the attack passes off recovery of power is usually simultaneous in arm and leg. He thinks there is little if any loss of power in the left face; it feels as though it were being stretched or dragged up, but it is not so powerless as the limbs.

Sequelæ: Usually has headache (temporal and frontal) after the fit, and this may last a whole day. There is no sickness or vomiting.

Patient thinks that the left side, arm especially, is becoming rather weak, but in any case the degree is slight. Otherwise, feels perfectly well.

State on admission: No indication of abnormality in circulatory, respiratory, or alimentary systems.

Cerebral: Attention, memory, intelligence, excellent. Is not conscious of any mental change. Not emotional. Apart from fits, no headache or giddiness.

Speech, taste, smell, hearing, unaffected.

Cranial: No cranial tenderness.

2. V.A. 6/6 R. and L. No restriction of fields, rough test.

O.D.: L. appears normal, though rather high-coloured. R. definitely high-coloured; veins full.

3.4.6. No diplopia, no ptosis, strabismus or nystagmus. No ophthalmoplegia.

Pupils regular, equal, medium size; reactions normal.

5-12. No impairment observed in the function of these nerves. No facial asymmetry or weakness. No defect in phonation, articulation, or deglutition. No defect of palatal movement. Pharyngeal reflex present. No facial hypæthesia or anæsthesia. Tongue protruded straight.

Motor System: General muscular nutrition and development good.

There is some general flabbiness of the muscles more marked in the left arm than in the right. No particular difference in legs.

No incoordination of any kind. No involuntary movements. Patient is right-handed.

There is perhaps just a little relative weakness of the left arm as a whole. No difference in power of legs, which is good.

All voluntary movements good in range.

Sensory System: No objective changes of any kind detected. No astereognosis. No atopognosis. No loss or diminution in sense of position or passive movement. No diminution of muscle pain sense.

Reflexes: Abdominal and epigastric present and equal; brisk. Knee jerks, arm jerks, brisk; no apparent difference between the two sides. No ankle clonus. Double flexor response. No organic reflex impairment.

Description of Fits Observed by Myself.

(1) 12.40 p.m.—When I saw the patient he was lying on his right side, with his legs drawn up, right not so much as left, and with his left arm flexed at the elbow and extended at the wrist. The left foot was distinctly inverted and toes were pointing downwards. He said that at that moment the whole of his left side felt numb, including the lower part of his face and round the nose on the left side. He further declared that the sensation had come on when his legs happened to be drawn up, and that he could not move his left leg. When I tested him, he was unable to move the leg at all, and could move the arm only at the shoulder and elbow, and that very slightly.

Patient had no appearance of distress, no difficulty in speaking, no twitching, and was absolutely conscious and coherent the whole time.

Testing him quickly, it was seen that his arm was rather rigid at the shoulder and elbow, and that the wrist and fingers were quite flaccid. The resistance to passive movement seemed to be due to defective inhibition of antagonists, a phenomenon well seen in the left leg. The leg was resistant to passive movement, more proximally than distally, and immobile. His face on the left side was distinctly affected, especially in the lower division, for both volitional and emotional movements. His lower maxilla was "chattering" and he was unable to control this. He remarked then that his tongue on the left side, and the parts underneath his tongue, were numb.

Tested quickly for sensation, there was anæsthesia of the hand and wrist, and of the leg. The limits were undefined. There was astereognosis in the left hand, and also very considerable diminution of the sense of passive movement and of position. There was very little if any diminution in appreciation of painful stimuli. All the tendon jerks on the left side were exaggerated, but I failed to elicit an extensor response on the left. The abdominal reflexes were diminished on the left.

12.46.—By this time there was some return of movement in the arm, and also in the leg. He could slowly push his leg down, though he could not lift it off the bed. He was able to flex and extend at wrist and could move the fingers slightly. The position of the foot was maintained. In the attempt to move the leg, defective inhibition of antagonists was particularly well seen. Thus as he slowly extended, the hamstrings were seen and felt to contract spasmodically. The same thing was noticed in the gastrocnemius when he tried to dorsiflex the foot.

Spontaneous left patellar clonus commenced at this point, and when the left knee jerk was elicited there was prolonged patellar clonus. Ankle clonus was likewise obtained, but it was not so well sustained. There was a left flexor response at this stage. The numbness was gradually disappearing from leg and face, but it persisted in the arm and hand. When I was testing his sensation for touch, he remarked that the touch of my finger on his leg and arm and hand gave rise to a feeling of pins and needles at the spot touched.

The facial asymmetry was absent now, and his tongue was protruded straight, although it was tremulous. There was no defect of jaw movement. The jaw clonus had disappeared.

The loss of sensation to touch remained, and stereognosis was still inaccurate.

12.55.—In the attempt to close his hand, there was some irregular twitching of the forearm muscles, chiefly of the antagonists to the movement. Diminution of muscle sense was not now so marked. By this time the movements of his legs were normal in range but not of normal strength. He complained that where his limbs had been numb they were now cold, but to my hand there was no actual difference in the temperature of the two sides.

1.02.—Asked to hold his arms out it was seen that the left arm was oscillating very slightly and irregularly, but he was able to hold it as well as the right.

The reflexes on the left side were now distinctly less brisk than a few minutes ago, although patellar clonus was still obtained. On stimulating the sole of the left foot, there was flexion—though very slight—of the great toe, and extension of the small ones. Left abdominal reflex less marked than right.

There was still slight persisting anæsthesia of the fingers, left, but not elsewhere.

1.08.—All movements on the left were now good in range, but there was slight weakness generally at all joints. The anæsthesia had disappeared.

(2) *Patient again seen in a fit one week later.*—When I saw him, he was lying on his bed with his clothes on. He was perfectly conscious, a little pale, and his lower jaw was chattering incessantly. His left arm and leg were helpless. The arm was flexed at the elbow, and lying across his body. On request, he was unable to lift the hand or arm off his body. Neither limb was at all stiff or rigid; on the contrary they were completely flaccid. He

complained that all the feeling had left the limbs on that side, and that the numbness was extending to the left side of his face. The numbness and coldness began in the fingers and passed up the arm and down the trunk to the leg.

On the left side the jerks were unusually brisk; there was definite though not well-sustained ankle clonus. Also there was apparently a left extensor response, though this was difficult to be sure of; sometimes there was no movement of the toe in response to the stimulus. The left arm jerks were also exaggerated. Both abdominal reflexes were active at this stage.

A minute or so later there was slight return of power in the limbs, distal more than proximal, and more in arm than leg. At this point sensation was rapidly tested, when it was found there was no diminution of appreciation of pain on the left side, but considerable diminution of tactile acuity.

Topognosis also was tested, when there was seen to be definite preaxial localisation.

Patient then remarked that the numbness had spread to the inside of the left cheek, and under the tongue on the same side. The chattering of the jaw was less, but he said that he had a disagreeable feeling in his throat, as though he were "swallowing a sob."

His sense of position and of passive movement was then tested in the left hand, and it was found that when his third finger was moved he referred it to the first, his fourth to his third, his second to his second. The diminution in sensation continued.

When I was testing his sense of pain again, in the left hand, he said that a prick felt hot as well as sharp. His fingers still felt numb, but the numbness was passing from the rest of him. From this point his left grasp began rapidly to improve, but it was noticed that he could not sustain it owing to defective innervation of the synergic and defective inhibition of the antergic muscles.

A few minutes later the subjective sensations had almost disappeared, except in the fingers. The jerks remained very brisk. The power of the limbs was returning.

(3) *Six days later, a third fit was observed in part.* It had begun with a sensation "like a battery" starting in the left hand and running quickly up the left arm and down the left side into the leg and foot, as well as into the tongue and face (left), and this was succeeded by numbness in the same left-sided distribution. When I saw him, he said he had lost all power over the left limbs. I found that the left side of the face moved less well than the right, and that the tongue was protruded distinctly to the left. The left limbs were limp and useless; voluntary power was practically nil, though there were just the slightest movements possible of finger flexion.

While I was conducting this brief examination, the clinical condition changed before my eyes (as on previous occasions). The left limbs became rather rigid, the arm moved (involuntarily) into a position of slight flexion at elbow and adduction at shoulder while the fingers partially closed. At the same time the left leg flexed slightly at hip and knee, extended at ankle, and assumed a position of adduction.

Within a minute or two power commenced to return; movements of the left arm increased in strength and were associated with a sort of coarse tremor or oscillation of the left arm at the shoulder—an internal and external rotation of the upper arm in succession. Passive movement of the left leg evoked a kind of spasmodic alternation of extension and flexion at hip and knee through a very limited range.

The deep reflexes were exaggerated on the left side, but both plantars were in flexion, and both abdominals were present and active. Sustained ankle clonus was readily elicited on the left side.

Some ten minutes after the commencement of the attack the patient was rapidly recovering both movement and sensibility, and in a quarter of an hour altogether the above-described symptoms and signs had vanished.

The hyperæmic condition of the optic discs eventually changed into a slight papilloedema, and the question of operation was broached. The patient refused to consider this, and passed from my observation.

DISCUSSION.

The case illustrates excellently the occurrence of akinetic or inhibitory phenomena in succession to pronounced sensory discharge, and offers features of considerable importance in respect of cortical physiology.

In the first place, we note that the sensory aura follows strictly the march that is to be expected from cortical excitation, viz., from the fingers up the arm to the shoulder and neck, thence down the trunk to the leg and toes, and at the same time into the tongue and face. This aura by itself is sufficiently indicative of the cortical site of the physiological disturbance. Secondly, the outfall of motor function is found to vary according to the stage reached in the course of the seizure, the affected limbs being flaccid at the outset, and passing through a period of relative rigidity on the way to recovery. When the arm and leg were completely flaccid they were also powerless, and as tone returned so did a degree of volitional innervation, though the two processes did not advance entirely *pari passu*. It would seem that some degree of return of muscular tonus preceded the other. Of especial physiological interest is the fact, determined in each of the three personally observed fits, that during the process of recovery from the stage of absolute flaccid akinesia the limbs for a time exhibited the phenomenon of defective inhibition of the antagonists in a striking fashion. In other words, disarray of the normal interaction of synergists and antergists for the time being existed. This clinical manifestation has long been recognised as liable to occur in cases of hysteria with motor symptoms: physiologically speaking, hysterical disorders are initiated at cortical or transcortical levels. But defective antagonist inhibition is not confined to cases of hysteria. Considerable attention was given in my Croonian lectures to personal observations made in cases of chorea and of athetosis, which have proved that in these states Sherrington's law of reciprocal innervation is often in abeyance and that interruption of the orderly sequence of agonist and antagonist innervation is frequently found. In the same series of papers arguments were advanced in support of the view that such interference with reciprocal innervation is commonly of cortical site and is not observable at lower physiological levels, or only exceptionally so.

If this hypothesis is well-grounded then the appearance of the sign of defective antagonist inhibition in the course of akinetic Jacksonian epilepsy is particularly informative, for it throws light on cortical motor function from the clinical side, and incidentally substantiates the general contention. It cannot be seriously maintained that the motor symptoms of this case of mine have any other origin than the motor region of the cortex.

Actual loss, or diminution, of the deep reflexes was not observed at any point in the course of the attacks in this case. Possibly, had I tested them at the period of maximum flaccidity, it might have been otherwise, but as motor and sensory states were on each occasion first submitted to examination the

reflexes were reached only after the lapse of several minutes, and this may have been sufficient for a stage of diminution to have passed. However this may be, their exaggeration, the presence of ankle clonus and, on one occasion, of a probable extensor response, clearly point to infracortical release synchronous with cortical inhibition In this connexion the appearance of an extensor response during a phase of the ordinary major epileptic fit may be recalled. I have not myself been fortunate enough to observe definite loss of the knee-jerk in the course of general fits, but Gowers states it is sometimes abolished immediately at the close of the stage of convulsions.

The development of temporary motor paralysis, without spasmodic discharge, in the course of attacks of the above description, must be assigned to lowered activity of the motor centres involved, that is, to their inhibition, and it is legitimate to assume that the discharge in the corresponding sensory centres has somehow been the cause of this. The likelihood of this being the correct explanation is by no means remote. Under a variety of circumstances powerful stimulation or excitation leads to arrest of function. When a person is "thunderstruck," is "struck all of a heap," in consequence of some violent sensory stimulus, he may be for the moment incapable of movement—he is "paralysed." Reflex action can be interrupted or inhibited by strong painful cutaneous impressions. The only satisfactory way of explaining the cessation of the motor phenomena of a Jacksonian fit by ligature of the limb concerned—a procedure known to the ancients—is by assuming an action on sensory centres and inhibition of corresponding motor centres. In a recent paper on epileptic variants (see this JOURNAL, vol. viii, p. 223) I have cited a case in which a strong sensory discharge (olfactory) was succeeded by a trance-like state of motionlessness. It is known that a negative phase or refractory state of a given physiological mechanism or reflex arc follows excitation. Too loud a sound deafens; too bright a light blinds. We may readily conceive of a refractory or inhibitory phase developing in a motor mechanism if it is suddenly or rapidly assailed with violence by afferent stimulations of an exaggerated character. However antagonistic and mutually exclusive processes of excitation and of inhibition respectively appear to be, they must without doubt be held to be at the same time closely interrelated. The most recent evidence pointing in this direction is furnished by the elaborate investigations of Pawlow, who has conclusively proved how readily inhibition follows stimulation, and how, in respect of cortical function, the two processes are constantly and continuously interacting and superimposing, one on the other.

This being so, it would be curious indeed were phenomena of inhibition not to characterise one or other of the phases, one or other of the manifestations, of what for want of a better term we call epilepsy. Epilepsy cannot call into being physiological processes that do not exist; at the most it can only distort or caricature the processes of the individual concerned. As Hughlings

Jackson said, "the convulsion is a brutal development of the man's own movements." Analogously, an epileptic development may be a sudden inhibition of the man's own movements.

My chief purpose in discussing the inhibitory side of epileptic semiology has been to bring the latter into line with other recognised clinical states of which akinesia is a, perhaps the, prominent feature. Elsewhere I have recently examined at length the clinical symptoms and pathogenesis of so-called narcolepsy and cataplexy, and have stressed the fact that "sleep" is somewhat of a misnomer in view of the observed nature of the clinical syndromes. These are constituted, rather, in numerous instances, by a state of immobility without loss of consciousness, and in my opinion are often allied to catalepsy on the one hand, and to epilepsy on the other. Instead of rigidly enforcing such criteria as may appear to differentiate all of these, it has been my aim to emphasise their parallels and resemblances.

Short Notes and Clinical Cases.

REPORT OF A CASE OF POSTENCEPHALITIC RESPIRATORY DISORDER ASSOCIATED WITH CONDUCT CHANGES: APPARENT COMPLETE RECOVERY.

By R. G. GORDON, Bath.

In the last number of this Journal Drs. Aldren Turner and Macdonald Critchley described their experience in regard to the prognosis of the disorders of respiration associated with epidemic encephalitis. Out of their series of 29 cases they describe eight as associated with conduct or psychotic changes, and of these one only appeared to have recovered in respect of both the respiratory disorder and the conduct derangement. It is not necessary to review the literature again here, but several writers have noticed this association and either have given a bad prognosis or where recovery has ensued have attributed this to some particular method of treatment. For example, Ely Jelliffe considers that psychoanalysis is particularly efficacious in this class of case. It may therefore be of interest to describe the following case, that of a patient who has been under close observation for 21 months.

Up to the age of 12 the patient had been a bright girl and had done well at an elementary school. She was the eldest of three children, with no family history of nervous or mental disease. Except for measles and whooping-cough, her own health had been excellent. She was regarded as exceptionally intelligent and was set to work for a scholarship to the High School. During the early summer of 1925 she became sleepless at night, complained of diplopia, and was noticed to have some ptosis. It was supposed however that this was simply due to the fact that she was overworking for her examination. She was allowed to go in for this, and obtained the scholarship. She apparently recovered from her sleeplessness and the other symptoms, which were rather indefinitely described after she came under observation.

Three months after starting work at the High School she was reported to be very nervous, inclined to be mischievous with other children and unable to keep up with the work of the class. It was advised that she should be taken away from school for a time and sent to the country. This could not be arranged and she remained at her home in the town.

In September 1926 she began to suffer from bouts of panting respiration, lasting from two to 30 minutes. During these attacks, which gradually became more violent and frequent, she seemed to lose all control of herself, to be very vague as to her whereabouts, and if contradicted or crossed would

hit out with her fists and throw any missile that came to her hand, from a knife to a teapot, at anyone who was in the room. In the intervals of the attacks she was uncertain, restless and bad-tempered, slept poorly and suffered from nightmares. No reflex changes were detected nor were there any signs of bodily disease.

By December 1926 the condition had become so bad that the family were worn-out and terrified. Her mother almost always had a "black eye," and her father, who was an ex-sergeant-major, scarcely ever appeared in public without obvious scratches and contusions. She was also very destructive, broke up the furniture and the windows of her home. Funds were obtained to send her to a small private residential school for backward and difficult children, to see whether a change of environment would have any effect. For the first two or three days she behaved better, but soon struck her governess and threatened the other children. As she was inclined to throw knives about, it was impossible to retain her at the school. At this time she was 13 9-12 in actual age and had deteriorated to a mental age of 10 4-12. This would certainly seem to have been a deterioration, as she could not have gained a scholarship with this degree of retardation.

The parents were now at their wits' end to know what to do and in order to deal with her promptly the only course seemed to be to take her to the Workhouse Infirmary. This was done but the parents refused to leave her there, and apparently the child was considerably impressed by nearly being taken to this institution, and from this point a gradual amelioration set in. She was seen regularly twice a week, but no treatment was given to her except the mildest psychotherapy, in the form of encouragement and exhortation. Certain obvious fears, such as those of large animals and of deep water, were discussed with her quite superficially, but by no stretch of the imagination could the treatment be described as psychoanalytic. All that could be said was that the physician obtained the confidence of the child and that she was prepared to make a certain amount of effort for him. To begin with, however, these efforts at control had no effect on the respiratory tic, which always retained the same form of violent panting.

It is difficult to describe any stages in the recovery which has continued gradually until the present time. She has now lost all trace of respiratory tic, her conduct is exemplary and she is acting as a pupil teacher in an elementary school and continuing her study of the piano with some success. A recent mental test shows that at the age of 14 11-12 she has a mental age of 12 8-12, a gain of 28 months during the interval of 14 months. Although this mental age is still below her actual age, it must be remembered that over the mental age of 12 the tests are much less reliable and many so-called normal adults fail to score much higher marks. The recovery however is distinctly striking. She sleeps and eats well and except for a slight degree of nervousness and excitability with strangers cannot be regarded as in any way abnormal.

In view of the typical sequelæ of respiratory and conduct disorder and the definite history of derangement of sleep and interference with co-ordination of eye muscles, there seems little doubt that she suffered from a mild attack of encephalitis lethargica. The prognosis of conduct disorders is generally held to be unfavourable, as is that of respiratory disorders: this is seen in both Jelliffe's series and that of Turner and Critchley. The apparent complete recovery of this case is therefore noteworthy. It is further to be observed that the recovery seems to have been almost entirely spontaneous, for the psychotherapy which she received was scarcely worthy of the name.

Editorial.

ON TREATMENT.

A COMMON reproach, almost indeed a gibe, levelled against neurologists is the barrenness of the field of treatment as compared with that of diagnosis. With the general truth of this criticism we cannot pretend to quarrel, if we except the syphilitic affections of the nervous system. It should, however, not be forgotten that the word treatment is not synonymous with cure. If it were so, the therapeutic activities of the whole medical profession would indeed be seriously curtailed. And although cure in the strict sense of the term may often be an impossible ideal, yet treatment still remains, symptomatic treatment upon the successful results of which much of the comfort and well-being of the patient may depend. There are many conditions, perhaps the results of some pathological process rather than the persistence of an active lesion, where carefully designed treatment may prove of great value, even although we may not be able to affect the essential cause of the symptoms. Again, there may be certain symptoms or complications which call urgently for relief, where the underlying cause is still present and still incapable of removal. Spasticity of the limbs and pain may be selected as examples.

It is well recognised that excessive hypertonus of different muscle-groups in the limbs may be found in many different conditions where a very fair amount of voluntary motor power is still retained, and in such cases the essential disability may really be due almost entirely to the muscular hypertonus. This is often spasmodic rather than truly tonic in character. The flexor spasms of many forms of paraplegia, the adductor and equinus spasms found so commonly in the infantile hemiplegias, are cases in point. There are, again, many varieties of old-standing intrinsic diseases of the spinal cord, such as disseminated sclerosis, and spastic paraplegia the result of old injuries, where the spasm is the predominating feature in the disablement. Realising only too well the incurable nature of the underlying lesion, do we make adequate efforts to treat its results? Too often, we fear, are such cases simply dismissed as

incurable. The orthopædic surgeon is often called upon to treat the deformities arising from the cerebral palsies of infancy or childhood just as he is to correct those due to the ravages of poliomyelitis. Do we make enough use of his services in other types of nervous disease?

Carefully designed operations, especially those of the Stoeffel type, may do much to relieve the spasmodic hypertonus which is often a marked feature of the chronic forms of disseminated sclerosis or of old cases of myelitis or old injuries of the spinal cord. Even if we have no guarantee that the pathological process is no longer active or that fresh developments may not arise later, the progress of the disease may be so slow and so essentially chronic that such measures of treatment are fully justified. Unfortunately the operation of sympathetic ramisectomy has not fulfilled the high hopes of success with which its advent was at first greeted: else the neurological surgeon would have had a very wide increase in his field of action. Surgical operations apart, however, there are other and minor methods which may relieve temporarily this disagreeable and disabling symptom of spasticity. Of such, heat is a simple and valuable example, as has been pointed out in this JOURNAL.*

Turning to the relief of pain by measures other than the mere administration of drugs of a sedative or narcotic type we may again ask ourselves whether we may not be more bold in invoking the assistance of our surgical colleagues for the treatment of this symptom also. The intractable pains of certain cases of tabes dorsalis or of certain rare forms of syringomyelia will at once occur to the mind as examples, as may also those distressing cases of malignant disease of the spine or indeed of malignant disease anywhere in the body where pain is a serious complication. Though in some cases the operation of division of the posterior roots, rhizotomy, has been attended by success, we have greater hopes of the wider application of the operation of chordotomy, where the anterolateral part of the spinal cord is divided and with this the upward path of the pain fibres. We venture to think in this field there will be ample scope for the beneficent activities of the rapidly increasing class of neurosurgeons, albeit they may not achieve that ideal, so dear to the surgical heart, of a radical cure.

The relief of symptoms, however, is not the prerogative of the surgeon alone. Ataxia is a symptom which, when it occurs in the more chronic forms of nervous disease, is often capable

* R. G. Gordon, vol. iii, 1922, p. 47.

of great amelioration by simple methods of re-education. With the modern methods of treatment by salvarsan, bismuth, etc., the technique of the now somewhat old-fashioned Frenkel's exercises in tabes has been almost relegated to the limbo of forgotten and discredited methods, and yet it is one capable of yielding excellent functional results. And amongst the multitude of different deformities resulting from poliomyelitis the value of re-educational muscular movements can hardly be exaggerated. It is indeed a method of treatment often of far greater importance than the conventional prescription of electricity and massage, beyond which in only too many cases no further suggestions have ever been made. The careful re-education of the aphasic is a labour which is seldom undertaken in any really conscientious manner and yet what greater boon can treatment confer on any patient than help in the recall of speech ?

It is time, therefore, that we should take steps to remove the reproach that there is no treatment in neurology. Every case, no matter how incurable in its essence, should receive its own special consideration from the standpoint of treatment, with no regard to the pessimist who would always decry the usefulness of symptomatic treatment. Those who, impatient of any method of treatment short of real cure, do not honestly and carefully treat symptoms do their patients little service and their profession little good ; they play only too often with the hands of the charlatan, who is maybe less critical of the ultimate value of the methods he employs.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[174] **On neurobiotaxis.**—C. U. Arien Kappers. *Acta Psychiat. et Neurol.*, 1927, ii. 118.

This paper is a report of a lecture given before the Neurological Society of Kopenhagen. It outlines the main features of the author's well known law of neurobiotaxis, formulated in 1907 and made familiar to English readers through recent publications (*Brain*, 1921). Neurobiotaxis is an extension of the law of mental association applied to the anatomy of the central nervous system. In general terms it means that structures of the nervous system are arranged according to the impulses which pass through them: furthermore, that structural propinquity results from simultaneous activity of discrete functions. The general principle is supported by numerous illustrations, which suggest that the "teleogic character of the intrinsic structure of the brain coincides with neurobiotactic causality."

M. C.

[175] **The relation of the cerebellum weight to the total brain weight in human races and in some animals.**—C. U. A. Kappers. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 113.

The literature is reviewed and the author's own observations on animals of various species and orders are discussed. From these it appears that the size of the cerebellum, especially of the hemispheres, depends on the use of the limbs, the largest hemispheres being found in those animals which require most careful and exact inhibition of the antagonists during the activity of the agonists.

R. G. G.

[176] **The columnar arrangement of the primary afferent centres in the brain-stem of man.**—W. Freeman. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 1.

Considering the brainstem in the same way as is commonly employed in the investigation and description of the spinal cord, the author lays it down that the fifth, seventh, ninth and tenth cranial nerves are similar in construction to the spinal nerves: each innervates a certain cutaneous area with general exteroceptive cutaneous sensation, and a certain area of mucous membrane with general interoceptive sensation. Each supplies motor fibres to muscles originating from the visceral motor system which once surrounded the alimentary

canal. Each carries proprioceptive fibres from the muscles of its area of supply and segmental reflex fibres. The course of these fibres is traced, and when the location of columns is compared at various levels it is seen that a constant relationship is maintained, practically identical with that observed in the spinal cord. The findings in the spinal cord can be applied directly to the brain-stem, making allowance for the overgrowth of certain components of some nerves and others of other nerves. Many of the structures cease to have an obscure and unusual significance and when disguise is stripped off become much more like the same structures in the spinal cord with whose appearance we are familiar.

R. G. G.

[177] **The role of the dorsal roots in muscle tonus.**—S. W. Ranson. *Arch. of Neurol. and Psychiat.*, 1928, xix, 201.

Ranson considers that in spite of all the work which has been done to determine its nature and cause, muscle tonus still presents an unsolved problem. Any theory to explain it cannot be considered satisfactory unless it provides a mechanism for maintaining posture without fatigue and with a minimum expenditure of energy, by a type of contraction which is more or less free from action currents. Two theories have been advanced in explanation of the part played by the dorsal roots. Of these, the proprioceptive reflex theory of Sherrington is the one generally accepted: but the theory of the antidromic conduction of tonic impulses in the dorsal root, advanced by Trzecieski and Frank, has much in its favour and must be given careful consideration. The author's experiments show that contracture, a permanent shortening of the extensor muscles which persists after section of the motor nerves or after the death of the animal, not infrequently develops in muscles deafferented by section of the dorsal roots proximal to the spinal ganglia.

Decerebrate rigidity disappears in a leg immediately after section of its sensory fibres in the dorsal roots. This is to be attributed in part to shock. If, after section of the dorsal roots, an animal has been allowed to recover and all resistance to passive flexion has disappeared before decerebration, decerebrate rigidity is slow in developing in the deafferented limb. But after the lapse of time, varying from thirty minutes to several hours, a tremulous oscillating type of rigidity develops, owing to rapidly succeeding contractions of the extensor muscles. Tonic neck and labyrinthine reflexes occur in the deafferented limb and are usually brisker and more extensive than on the normal side.

While section of the dorsal roots does not prevent the development of decerebrate rigidity, it deprives it of one of its chief characteristics, namely, plasticity. This plasticity, which is responsible for uniform stiffness and which causes a limb to hold a posture passively imposed on it, is dependent on integrity of the dorsal roots. According to the theory of muscle tonus that one holds, one can explain this plasticity as a steady contraction called forth

by afferent impulses from the tonic muscles themselves, or as due to special tonic impulses from these, or as due to special tonic impulses travelling antidromically over the dorsal roots.

R. M. S.

[178] **Reflexes in apes.**—G. D. Aronovitch. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 457.

The author investigated the presence or absence in apes of the reflexes which are familiar in man. These were found to be well established except in the case of the abdominal reflexes, which were not found. The explanation is that that reflex is associated with the erect attitude, since the unsupported abdominal muscles require a much more active tone than is the case in animals which are only semi-erect.

R. G. G.

[179] **Studies on the cerebral pulse** (Études sur le pouls cérébral).—J. Tinel. *L'Encéphale*, 1927, xxii, 229.

Tinel has studied the cerebral pulse in patients who had had craniotomies performed for various causes. After investigating the various fallacies attendant on this somewhat indirect method of study, he noted the effect of various psychical and physical changes and of some drugs. Slight emotional stimuli, pain and attention produced varying results, but on the whole slight stimuli produced vasodilatation, more severe and prolonged stimuli vasoconstriction and very violent stimuli almost always vasodilatation. Stimulation of the solar plexus produced vasoconstriction, while the oculocardiac reflex caused vasodilatation. Among pharmacological agents adrenalin very markedly, and strychnine and caffein to a less degree, increased the abruptness with which vasoconstriction and vasodilatation took place, whereas tartrate of ergotamine damped down these changes or made them disappear completely. Amyl nitrite and formalin caused vasodilatation. Inhalation of oxygen caused a remarkable degree of vasoconstriction, and the same effect could be obtained by hyperpnœa. That the latter was due to excess of oxygen was proved by its absence when forced breathing was performed in an atmosphere overcharged with CO_2, as by re-breathing in and out of a bag. The author draws three conclusions from his experiments:—

(1) The cerebral circulation is under nervous control, although it is not directly affected by adrenalin.

(2) Vasoconstriction and vasodilatation have not any direct or necessary relationship to mental activity or psychic state.

(3) In patients suffering from postencephalitic crises these can be brought on at will by any agency causing vasoconstriction and dissipated by agencies causing vasodilatation.

The author discusses the possibility that other, so-called hysterical, states may be due to angiospasm of the cerebral vessels.

J. G. Greenfield.

[180] **A comparison of the tension of the retinal arteries and of the cerebrospinal fluid** (Tension rétinienne et tension du liquide céphalorachidien).—H. Claude, A. Lamache and J. Dubar. *L'Encéphale*, 1927, xxii, 1.

The systolic and diastolic pressure of the retinal arteries can now be studied by the tonometer of Bailliart, in the use of which ophthalmoscopic examination of the retinal artery on the disc during compression of the eyeball shows pulsation beginning at diastolic pressure and ending at systolic pressure. The authors compared the results obtained by this instrument with the blood pressure and cerebrospinal fluid pressure. The normal pressures in the retinal artery vary from 600 to 700 mm. of water for the systolic and from 300 to 350 mm. for the diastolic. As a general rule it was found that the diastolic retinal pressure bore a close relationship to that of the cerebrospinal fluid and varied with it when it was artificially raised or lowered. When the cerebrospinal fluid pressure was normal the retinal diastolic pressure varied with that of the general arterial system. But sometimes the retinal pressure was found to be unexpectedly high by comparison both with the arterial and the cerebrospinal fluid pressure. In some cases this appeared to be due to emotional stress at the time the retinal pressure was taken. In other cases, as in general paralysis, it was probably due to some local blockage to the outflow from the retinal veins or lymphatics. The authors recommend the examination of retinal pressure both in cases of suspected brain tumour where lumbar puncture is contraindicated, and in cases in which lumbar puncture has proved the existence of raised intracranial pressure when it is considered advisable to watch the effects of hypertonic solutions in reducing this.

J. G. Greenfield.

NEUROPATHOLOGY.

[181] **Pathologic changes in Huntington's chorea.**—C. R. Dunlap. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 867.

The late Dr. Charles Dunlap has in this paper contributed a minute and painstaking account of the pathological changes in seventeen cases of Huntington's chorea. It would be hazardous to attempt a summary of the many points discussed, but Dr. Dunlap's views seem to amount to this, that in pure and well developed Huntington's chorea there are constant lesions of definite type in the corpus striatum and in the cerebral cortex and marrow which are characteristic enough to make a postmortem diagnosis of Huntington's chorea at least probable if not certain, for the peculiar combination of cortical changes and basal nuclear lesions is not reproduced in any other disease.

R. M. S.

[182] **The neuropathological findings in a case of acute Sydenham's chorea.**—L. H. ZIEGLER. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 273.

THE study of a case under the care of the author and of the literature shows that this syndrome is associated with chromatolysis of practically all cells of the central nervous system, with swelling of nuclei and excentric displacement: destruction of some neurones especially those of the sixth nerve and calcarine cortex, where glia cells were much proliferated; neuronophagia, fatty deposits in the large cells of the motor cortex and globus pallidus; fat in the perivascular spaces and petechial hæmorrhages in a small area near the dorso-medial aspect of the restiform body of the medulla. Recent reports show changes in the corpus striatum similar to those found in the lesions of epidemic encephalitis. Some of the elements of the brain may be less resistent to toxins or infections, or the latter may have a selective action. Though recovery takes place awkwardness of certain movements which persist suggests an inherently inferior motor system, or maybe a residuum of the disease.

R. G. G.

[183] **The histology of post-vaccinal encephalitis.**—J. R. PERDRAU. *Jour. Pathol. and Bacteriol.*, 1928, xxxi, 17.

PERDRAU has examined the lesions in the brain and spinal cord of three fatal cases of post-vaccinal encephalomyelitis. The onset of the disease was seven, nine and twelve days respectively, after a normal vaccination. He lays particular emphasis on the nature of the lesion and confirms McIntosh and Turnbull in finding perivascular zones of softening or, more correctly, of demyelination with relative preservation of the axis-cylinders. The cellular reaction in these areas is characterised by proliferation, mitosis and karyorrhexis in wandering cells, apparently of endothelial origin, and by early changes in the microglia leading to the formation of granulo-adipose cells. Demyelination was complete even in a case which ended three days after the onset of symptoms.

Comparing this type of lesion with that of poliomyelitis and encephalitis lethargica, he finds that although they resemble one another in the perivascular cellular exudate (lymphocytes and plasma-cells), yet post-vaccinal encephalitis is sharply distinguished by the tendency to perivascular demyelination. This lesion is in fact histologically identical with that found in early cases of disseminated sclerosis, and similar to that in some early cases of Schilder's encephalitis. It is also similar to that described in the published accounts of encephalitis or myelitis following smallpox, measles, and antirabic inoculations. The possibility that paralysis occurring after varicella and typhoid fever has a similar pathological basis is also suggested. He is inclined to the view put forward in 1925 by Bastiaanse, that the essential virus causing this form of encephalomyelitis is not identical with that causing the eruptive fever, but rather is an independent virus stimulated to activity by the febrile disease. In this connection he refers to the view put forward thirty years ago by Sottas

and Pierre Marie that disseminated sclerosis owes its origin to an attack by one of the eruptive fevers. While this view is not generally held at the present time, the similarity of the early stages of the lesion in disseminated sclerosis, Schilder's encephalitis and post-vaccinal encephalitis suggests a resemblance in the etiological agent, and leads to the hope that the discovery of the virus of one may throw light on that of the others.

J. G. GREENFIELD.

[184] **Zoster myelitis. The acute tephromyelitis of herpes zoster** (La myélite zostérienne. La tephromyélite de l'herpes zoster).—J. LHERMITTE and M. NICHOLAS. *L'Encéphale*, 1927, xxii, 245 and 313.

THE authors report a case of herpes zoster affecting the skin area supplied by the first to the fourth left cervical segments and followed by severe neuralgia. The patient died of bronchopneumonia eight weeks after the onset of the herpes. In addition to the typical lesions in the dorsal root ganglia they found inflammation and necrosis of the posterior horn on the left side of the second and third cervical segments of the spinal cord. In a wide survey of the literature they found that this histological finding was by no means uncommon. Not only so, but in many cases clinical evidence of myelitis was afforded by increased tendon jerks or absent abdominal reflexes on the side of the eruption, or by wasting of muscles supplied by nerves other than those implicated in the cutaneous eruption. For example, oculomotor palsy might be associated with facial herpes, or facial palsy might accompany an eruption on the neck or chest. It is not, therefore, possible in all cases to attribute the accompanying paralysis to pressure on the motor root by the swollen dorsal root ganglion.

With regard to the vexed question of the pathogenesis of the skin lesion, the authors refer to the work of several authors, in particular Lehner, who found that the skin lesions even from the earliest stages showed the characteristics of an inflammatory dermatitis. They consider it as proved that the virus of zoster attacks directly both skin and dorsal root ganglia, that it frequently reaches the spinal cord, and that it may even spread within the central nervous system.

J. G. GREENFIELD.

[185] **Metastatic carcinoma of the central nervous system.**—N. W. WINKELMAN and J. L. ECKEL. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 1.

TWENTY-THREE cases are presented and the literature is reviewed. The authors point out that the breast and prostate seem to predominate as the seats of primary tumour sending metastases to the central nervous system. These metastases may be in relation to the dura, to the nervous tissue itself, or within the perivascular spaces and blood-stream. Metastatic tumours do not differ from primary tumours of the central nervous system in their clinical manifestations. A history of operations for tumour especially on the breast or prostate is of the greatest importance in diagnosis, especially in view of the fact that metastases may recur years after such operations.

R. G. G.

[186] **Colloidal gold reactions with spinal fluids contaminated with blood.**—H. G. MEHRTENS, H. A. WYCKOFF, and R. M. DAVIS. *Arch. of Neurol. and Psychiat.*, 1928, xix, 296.

SPINAL fluids which are contaminated with blood, but from which the cells are removed by centrifugalisation before hæmolysis occurs, produce a colloidal gold curve altered in intensity only: the type of the curve remains the same.

Spinal fluid from patients with general paralysis made normal by treatment, on contamination with blood plasma, shows a tendency to revert to the former general paralytic type of curve. The original colloidal gold curve of spinal fluids experimentally contaminated with the patient's blood can be approximately reconstructed, provided hæmolysis has not occurred. There appears to be reason to believe that similar reconstruction can be made of curves of spinal fluids accidentally contaminated.

R. M. S.

[187] **The permeability of the haemato-encephalic barrier in nervous and mental diseases** (Sur la perméabilité de la barrière nerveuse centrale dans les maladies mentales et nerveuses).—S. BAU-PRUSSAK and L. PRUSSAK. *L'Encéphale*, 1927, xxii, 176.

OF the many tests for increased permeability of the barrier between the blood and the central nervous system, the bromide test of Walter and Hauptmann appears to be the most delicate, as by it we can estimate not only slight degrees of increase but also diminution of the permeability of the barrier. This test has been applied recently to the diagnosis of mental diseases, since the work of Stern and Gautier showed that any substance which can pass from the blood into the cerebrospinal fluid will also, under the same conditions, pass into the tissues of the brain, and since Monakow demonstrated certain changes in the choroid plexus in cases of mental diseases such as schizophrenia. The test is performed as follows: The patient is given for three to five days a daily dose of sodium bromide corresponding to 30 gr. for every five stones of body weight. On the day following the last dose specimens of blood and cerebrospinal fluid are collected at the same time and the percentage of sodium bromide in them estimated by a colorimetric method. (Precipitate the protein from an aliquot quantity of cerebrospinal fluid or blood plasma by trichloracetic acid. To 1 c.c. of the filtrate add 0·2 c.c. of ·5 per cent. gold chloride, and compare in the colorimeter with a standard sodium bromide solution similarly treated.) The percentage of bromide in the blood plasma, divided by the percentage in the cerebrospinal fluid, gives a quotient of permeability which is normally between 2·9 and 3·3. (The lower the quotient the greater the permeability: v. this JOURNAL, viii, 270.)

The authors investigated 108 cases by this test. These fell into five main groups. (1) Mental disease including general paralysis, 26 cases. (2) Organic disease of the brain including meningitis, 39 cases. (3) Affections of the spinal cord, 26 cases. (4) Affections of peripheral nerves and inorganic nervous

diseases, 14 cases. (5) General diseases, three cases. Of the mental diseases general paralysis was usually associated with an increased permeability, but after malarial treatment this became normal. Schizophrenia was often associated with a lowered, and never with a raised permeability. In arteriosclerotic and senile dementia a raised permeability was present. In acute meningitis and in syphilitic meningitis the permeability was increased, but not in encephalitis lethargica or in subarachnoid hæmorrhage. In tumours of the cord the permeability was increased in the lumbar fluid. In epilepsy, hysteria, affections of the peripheral nerves and in general diseases not affecting the nervous system the quotient fell within normal limits. In seven cases where the cisternal fluid was compared with the lumbar fluid, it was always found to have an abnormally low percentage of bromide, indicating that the normal values as given by Walter only hold true for the lumbar fluid. This observation indicates that it is necessary to restandardise the test on the cisternal fluid if it is to be applied to the diagnosis of mental diseases.

J. G. GREENFIELD.

[188] **The pathology of amyotonia congenita.**—ROY R. GRINKER. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 982.

AMYOTONIA congenita has been linked by some to the myopathies and by others to the infantile type of progressive muscular atrophy, and the pathological reports show considerable divergence. In the case reported by Grinker the symptoms were clearly those of amyotonia and the essential pathological changes were entirely within the confines of the lower motor neurone. The absence of glial reaction, scar-formation or vascular change suggests that this was not an active process, and certainly not an inflammatory or toxic condition. The simple paucity of cells of the anterior horn and the presence of cells which were either small, rounded and poor in chromatin, or long and fusiform, suggested a developmental defect. The muscle changes were most marked in the skeletal muscles. There were no fatty or degenerative changes, but simple muscular fibres in miniature containing all the markings of the normal muscle. Hypertrophic fibres were not noted, although they have frequently been described by others. The smooth muscle did not share in the process, and correspondingly the cell group of the lateral horn was well developed.

The author concludes that amyotonia congenita and the Werdnig-Hoffmann type of progressive muscular atrophy are identical as far as the pathological changes in the muscle and the site of the changes are concerned, but differ as to the type of lesion in the cord. In one a paucity of cells of the anterior horn with the presence of abnormal cells is found; in the other actual degeneration of the ganglion cell and neuronophagia. The fundamental difference in the two groups is probably only a matter of the time at which the ganglion cells are affected. All the infantile degenerative muscular atrophies may be classed according to this pathological criterion and there is no definite clinical differentiation between them, each type merging into the other. R. M. S.

[189] **Lesions of nerves in experimental lead poisoning** (Lesions des nerfs dans l'intoxication saturnine expérimentale, etc.).—J. M. DE VILLAVERDE. *Travaux du lab. de récherches biol.*, 1926, xxiv, 158, 267.

THE author has set out to examine the results of lead poisoning on the peripheral nervous system by the aid of Cajal's silver impregnation methods. He found that the nerve suffered as a whole, neurofibrils, axoplasm, myelin sheath and sheath of Schwann all being affected. This led to some curious anomalies in the microscopic appearances, for axis-cylinders might remain dead for some time without being broken up and removed by phagocytic activity either in the Schwann or other cells. Similarly, even when an axis-cylinder appeared to be destroyed very little attempt at regeneration took place, and this was always abortive. This might be attributed to some extent to the lesions found in the anterior horn cells, which are described as shrunken and as nowhere presenting the phenomena of axonal reaction. The motor endings on the muscle fibres were also studied and found to present changes similar to those found by Boecke after nerve section, but of less degree. There was an early loss of the network of Boecke in the end-organ and an early degeneration of fine intracapsular branches, with varicosity of the terminal part of the muscular twig. The poison therefore appears to attack all parts of the neurone simultaneously and independently, a fact which explains the slightness of the recovery which may be expected in severe cases. The paper is rich in microscopical detail and illustrations and contains a full bibliography of previous work on the subject.

J. G. G.

SENSORIMOTOR NEUROLOGY.

[190] **Subarachnoid hæmorrhage from a medico-legal point of view.**—WILLY MUNCK. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 484.

THE author points out that an isolated subarachnoid hæmorrhage may be the cause of sudden death and describes nine such cases. He remarks that in the absence of a reliable history it is practically impossible to determine whether the hæmorrhage is spontaneous or due to trauma, or to ascertain its starting-place.

R. G. G.

[191] **Intraventricular hæmorrhage.**—I. J. SANDS and M. LEDERER. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 360.

THREE cases are described and the authors point out that premonitory symptoms of intraventricular hæmorrhage are usually absent though the patient may show signs of cerebral arteriosclerosis or may be suspected of cerebral aneurism. An acute onset of cranial symptoms with the early appearance of coma, persistent blood-stained spinal fluid, the presence of repeated tonic spasms of the entire somatic musculature with the absence of classical signs of paralysis, should lead to the diagnosis of intraventricular hæmorrhage.

R. G. G.

[192] **Occlusion (embolic) of the posterior inferior cerebellar artery confirmed at autopsy, with pains and hyperalgesia to cold** (Un cas d'occlusion (par embolie) de l'artère cérébelleuse postérieure inférieure verifée à l'autopsie, avec des douleurs et de l'hyperalgésie au froid).—KNUD WINTHER. *Acta Psychiat. et Neurol.*, 1927, ii, 294.

A CLINICOPATHOLOGICAL account of a fatal case of Wallenburg's syndrome in a spinster of 64. The chief points of interest lay in the appearance of spontaneous pains and dysæsthesiæ in the affected side of the face, some three months after the ictus. She described a sensation of wetness or of "rain" behind the left eye. Objective examination showed an analgesia over the left face; there was an impaired appreciation of warmth over that area and a complete failure to recognise cold objects. Two months later burning pains appeared in the right arm, and an altered response to thermalgesia tests became apparent. She over-reacted intensely to contact with cold objects applied to the left face or right side of the body: the resulting sensation was one of pain; the actual *coldness* of the object could not be recognised at all. Ability to detect pain and warm stimuli was impaired but not lost. Winther remarks that the over-reaction to cold and the presence of spontaneous pains recall vividly the features of the thalamic syndrome, but in this case the pathological evidence places the lesion well below that level. This particular manifestation is on a par with the central pains due to focal lesions within the pons as described in syringobulbia. The author remarks on the similarity to the manifestations of causalgia and queries whether the sympathetic system may not share in the causation of the pain. He remarks on the presence of a Claude Bernard-Horner syndrome in his case, although this latter may exist with the presence of pain and dysæsthesiæ.

Another interesting clinical feature of this particular case lies in the symptom of obstinate hiccough.

M. C.

[193] **Thrombosis of the spinal arteries** (La thrombose des artères de la moelle épinière).—HELGE VEDSMAND. *Acta Psychiat. et Neurol.*, 1927, ii, 177.

THE role of primary vascular changes in the production of myelomalacia is imperfectly understood. In numerous cases of syphilitic myelitis it is claimed that the vascular changes are primary and not the result of an inflammatory process. The clinical manifestations of spinal arteriosclerosis, however, are almost unknown, except for a proportion of those cases spoken of as senile paraplegia. Vedsmand gives details of eight cases which he has diagnosed as instances of thrombosis of the spinal arteries: in only two, however, was autopsy verification possible. These two cases were characterized pathologically by complete necrosis of the dorsal cord with ascending and descending degenerations. Vascular occlusions were demonstrable and there was no

evidence of an inflammatory process. All of the eight cases were characterized clinically by a rapid onset of flaccid paraplegia, preceded for a varying period by pains and dysæsthesiæ in the legs. Sphincter control became lost, and sensation grossly impaired over the paralysed members. Superficial and deep reflexes were in abeyance. Except for the two cases which ended fatally, more or less complete recovery was the rule. Syphilis was admitted by six of the patients, although the blood and spinal fluid reactions were not strongly positive.

M. C.

[194] **The evolution of an encephalitic dystonia into a hypertonic-akinetic syndrome resembling Wilson's progressive lenticular degeneration.**—S. Brock and S. Katz. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 460.

A case of encephalitis is described in which observations were carried out at intervals of three years. The chief feature of the disease on the first examination was dystonia with marked involuntary movements. Three years later there was extreme rigidity and abnormal posture with no involuntary movements. The authors attribute this alteration to progressive changes in the lenticular nuclei and compare it to the clinical features found in Wilson's disease.

R. G. G.

[195] **Encephalitis after vaccination.**—D. Wiersma. *Acta Psychiat. et Neurol.*, 1927, ii, 167.

An interesting and valuable contribution to our knowledge of the neurological complications of vaccinia. The author has had under observation eleven cases of encephalitis occurring at intervals from nine to 19 days after vaccination. Convulsions occured in five of the cases: in three there were definite palsies; meingeal symptoms were seen in four: trismus in two: in seven cases a Babinski response appeared. Muscle tonus was diminished in all the cases and frequently the tendon jerks were unobtainable. Death occurred in five of his cases, but in two only was an autopsy allowed. Contrary to the opinion of Lucksch, Wiersma agrees with most other writers in regarding vaccinial encephalitis as distinct from encephalitis lethargica: the anatomical picture, too, differs in several important particulars. Thus in vaccinial encephalitis the perivascular infiltration often contains polymorphs, and the white matter is affected more than the grey. Wiersma discusses at length Bastiaanse's theory that a latent virus in lethargic encephaïtis is activated by the vaccination.

Although there is a certain amount of suggestive clinical and pathological evidence, the possibility of an association between vaccinial encephalitis and polioencephalitis is not discussed.

M. C.

[196] **Encephalitis periaxialis concentrica.**—JOSEPH BALO. *Arch. of Neurol. and Psychiat.*, 1928, xix, 242.

A LAW student, age 23, became ill with aphasia and agraphia; then followed weakness of the inferior branch of the facial nerve and disappearance of the cremasteric reflex on both sides and of the abdominal reflexes on the right side. Later, a hemiplegia of the right side, loss of sphincter control and a total aphasia developed. Before death, tonic spasms occurred in the right arm and leg; trismus developed, and the patient lost consciousness. Simultaneously, the right knee reflex and the achillis reflex became exaggerated, and the Babinski phenomenon appeared on the right side. At the beginning, the fundi were normal; later, optic neuritis developed. There was moderate elevation of temperature at this stage. The Wassermann reaction was negative, and a slight leucocytosis was present. Examination of the cerebrospinal fluid did not reveal any pathological condition. Four months after the onset of the disease an operation was performed, but no cerebral tumour found. The patient died shortly afterwards from intracranial hæmorrhage.

Postmortem examination did not reveal any pathological conditions in the internal organs. In the white matter of the brain foci were found varying in size from that of a lentil to that of a pigeon's egg. Some resembled gray softening; others were characterised by concentric layers composed of normal white matter alternating with layers of gray softening. In the latter fatty degeneration of the medullary sheaths was found. The most characteristic alteration of the neuroglia was the formation of giant glia cells. These neuroglia cells showed degeneration and were the subject of gliaphagia. Near the foci the periadventitial spaces were filled with products of degeneration, such as granule cells, lymphocytes and plasma cells. In areas of the brain widely distant from the pathological changes described above hyaline thickenings of the arterial coats were found. The cortex, basal ganglia and spinal cord were normal.

The changes about the vessels indicate the inflammatory character of the process. Syphilis, apparently, does not play a part in the etiology. Bacteriological examination did not reveal any micro-organisms. Intracerebral inoculation with material from the brain into rabbits did not transmit the disease. One would infer from this that the causative agent is a lecithinolytic ferment that exercises its action from different foci at different intervals. Among the diseases known to date, the condition in the case reported resembles acute multiple sclerosis and the encephalitis periaxialis diffusa of Schilder. The disease differs from Schilder's encephalitis because of its focal character. It differs from multiple sclerosis because in that condition both the brain and the spinal cord and both the gray and the white matter are affected. It resembles most the case of Barré, Morin, Draganesco and Reys and the third case of Marburg described in his work on acute multiple sclerosis. Balo suggests the term Leuko-encephalitis periaxialis concentrica for the pathological condition described in his paper. R. M. S.

[197] **Encephalitis periaxialis diffusa (Schilder).**—Georg Schaltenbrand. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 944.

A schoolgirl, age 14, was admitted because of a suspicion of appendicitis. When first examined she was apathetic and had a right-sided facial weakness and optic neuritis. During her sojourn in hospital she gradually developed choked discs and signs of extensive bilateral lesions of the brain, so that the probable diagnosis of encephalitis periaxialis diffusa was made. She died after an illness of three months' duration, in a state indicating practically complete disappearance of all cortical functions.

The postmortem examination of the body revealed nothing of importance pathologically. The brain, however, showed widespread softening and disintegration of the white matter of both hemispheres, the gray matter in general being spared. The process involved the corpus callosum and extended downward as far as the midbrain and the chiasma.

The outstanding feature of the less extensively involved areas of the spreading process was an abnormal filling of the blood vessels and a mucoid degeneration of the oligodendroglia, which proved to be identical with Penfield's acute swelling of the oligodendroglia. The areas of severe degeneration showed a complete destruction of the myelin, which was phagocytosed by scavenger cells of microglia origin. The axis cylinders were partially preserved and the neuroglia had hypertrophied in number and in size of the cells and formed a thick fibrous network. The perivascular spaces were enlarged and filled with scavenger cells and a few lymphocytes. In the regions of severest destruction there was degeneration also of the astrocytes into polynuclear forms without prolongations, dissolution of the rest of the nervous tissue, and formation of a scar of connective tissue. The engorgement of the vessels and the occasional perivascular hæmorrhages in this case suggest that the disease may be caused by an ultramicroscopic virus. R. M. S.

[198] **Centrolobar sclerosis of the brain** (La sclérose cérébrale centro-lobaire à tendence symétrique. Ses rapports avec l'encéphalite périaxiale diffuse).—C. Foix and J. Marie. *L'Encéphale*, 1927, xxii, 81.

The authors describe in detail three cases in which monoplegia, diplegia or quadriplegia came on in an acute or subacute manner, remained at a maximum for one or two months and then receded somewhat, leaving the patient severely crippled. The ages of the patients at the onset of symptoms were 18 years, 4 years, and a few months respectively. Death occurred at the ages of 28, 14 and 65. In none of the three was there any tendency to increasing paralysis or to mental deterioration after the initial attack. In all, however, the lesion in the brain was identical with that in Schilder's encephalitis in macroscopic appearance and differed only in the absence of any evidence of inflammation or of recent demyelination. There was in fact a neuroglial scar which replaced a large area of the white matter of one or both cerebral hemispheres, but spared the cortex, the subcortical zone of fibres and the basal ganglia.

The authors review the literature of Schilder's encephalitis, and show that the case of Pierre Marie and Foix (which appears to be the same as the first of the cases described in this paper) is the only one in which the disease was not progressive, although in the case of Claude and Lhermitte there were phases of slight regression of symptoms in the intervals between the phases of progression. There appears therefore to be a form of encephalitis, similar in its effects to that described by Schilder, but differing from it in clinical course. The onset is rather more acute (although it could scarcely be more acute than the loss of vision in two of the published cases of Schilder's encephalitis) : there is then a period of maximal paralysis lasting for several weeks or months followed by a period of slight regression of symptoms and permanent disablement. The extreme spasticity of the paralysed limbs is emphasized. In the third case here described a spastic club foot was the only sequel, but this remained unchanged for over 60 years. It is obviously impossible to distinguish the anatomical lesion from that of Schilder's encephalitis, since the only difference is one of age. It may be indeed that some of the more acute cases described as Schilder's encephalitis, if life had been prolonged, might have become stationary and would then have conformed to the type of Foix and Marie. The evidence given by this paper suggests in fact, if it does not prove, that Schilder's encephalitis, in some of its more acute forms, may be non-progressive and may in fact constitute the anatomical basis of some cases of spastic diplegia developing in infancy or childhood.

J. G. Greenfield.

[199] **Chorea of Huntington with endocarditis and polyarthritis.**—James F. Clancy. *Acta Psychiat. et Neurol.*, 1927, ii, 87.

A case is described of a female, age 69, with chorea of 10 years' standing. There was an indefinite family history of choreiform movements but none of mental disorder. At the age of five the patient developed endocarditis, and rheumatism at 17. One year before coming under observation she began to have fits without complete loss of consciousness. There was no severe dementia and orientation was good. Choreiform movements were severe and universal in distribution : speech was almost unintelligible. Two months before death she developed a subacute polyarthritis with fever. Autopsy revealed recent and old endocardial vegetations. Cross-section of the brain showed a marked shrinkage of the caudate nuclei on both sides, with some ventricular distension. The oral part of the caudate was the more affected : there was a numerical atrophy of cells and a glial increase. Some of the ganglion cells contained a yellowish pigment. Although the smaller cells of the neostriatum were most affected, it was thought that the larger ones were also altered. The globus pallidus was almost normal. In its anterior half the corpus callosum was small though well myelinated. There were very pronounced cortical changes, most marked in the frontal lobes and central gyri.

The polyarthritis and endocarditis are important factors which possibly played an exogenous role in the etiology by precipitating symptoms in an organism already weakened by hereditary influence. Connection between the chorea of Huntington and of Sydenham, a conception discussed in the older literature, is once more suggested.

M. C.

[200] **Cyst of third ventricle; complete destruction of infundibular region without any so-called hypophyseal signs** (Kyste du troisième ventricule, déstruction totale de la région infundibulaire sans signes dites hypophysaires).—L. FREY. *L'Encéphale*, 1927, xxii, 21.

THE case reported is an argument against the theory advanced by Camus and Roussy that the signs of so-called dyspituitarism are in reality due to destruction of the infundibulum and not of the pars posterior hypophyseos. A cystic tumour of the third ventricle had destroyed completely the infundibular region but had only compressed the pituitary body slightly. The only symptoms of dyspituitarism were a slight eunuchoid constitution and impotence. The case is interesting also in that the first symptom was trigeminal neuralgia. This persisted and proved to be due to extension of the tumour to the region of the Gasserian ganglion. Another symptom of interest was nystagmus on lateral deviation of the eyes, which was not due to any demonstrable lesion either in the brainstem or cerebellum.

J. G. GREENFIELD.

[201] **Segmental trophic œdema of cerebral origin.**—A. GORDON. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 381.

A CASE is described with a sudden onset of astereognosis of the left hand, probably due to the thrombosis of a cortical vessel in the parietal lobule. Later a localized œdema of the hand developed. Usual tests failed to disclose any signs of sympathetic involvement: medullary disease could also be eliminated, and the author thinks that the injury to the sensory cortex involved in the acute lesion was responsible for the production of the "main succulente."

R. G. G.

[202] **Syphilitic meningitis in infants and young children.**—J. W. AMESSE and W. W. BARBER. *Amer. Jour. of Syphilis*, 1927, xi, 544.

THE authors report four cases of meningitis in young children, in three of which the Wassermann reaction in the blood and spinal fluids was positive, but in the fourth negative. This last case was presumed syphilitic from the course of the illness and the absence of other etiological factors, and also from a suspicious history of parental nervous disease. The authors comment on the rarity of the condition, its clinical similarity to tuberculous meningitis and its prolonged course and unfavourable prognosis even with intensive antisyphilitic treatment.

J. G. GREENFIELD.

[203] **The pupils as an aid to the diagnosis in states of coma.**—W. C. Menninger. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 553.

The state of the pupils examined in 225 cases of complete coma is described. No very definite results are found and the author does not think that the pupillary condition is any help in the diagnosis of coma due to alcohol, diabetes, uræmia or carbon monoxide. In cerebral trauma or tumour the pupil tends to be dilated on the side of the lesion, while in pontine hæmorrhage the pupils are contracted. In fracture of the skull the light reflex tends to be abolished.

R. G. G.

[204] **A case of combined sclerosis in pernicious anaemia with polyneuritis, presenting the symptoms of a senile paraplegia.**—H. de Jong. *Acta Psychiat. et Neurol.*, 1927, ii, 105.

A woman of 69 gradually became weak in her legs over a course of years. On one occasion she sustained an ictus which was followed by further weakness in the legs and paræsthesiæ in the hands. Neurological examination revealed some weakness of the right face and tongue and arms. Both legs were paretic but especially the right one. The tone was increased: the ankle jerks could not be obtained, although the knee jerks were present. Both plantar responses were of the extensor variety. Some atrophy was present in the leg muscles and the electrical reactions were those of degeneration. Her tongue was red and somewhat painful; blood pressure 216–90; blood count:—reds 2,100,000; hæmoglobin 45 per cent., colour-index 1·38, leucocytes 11,000. There was some sensory loss over the legs. The indirect Van den Bergh reaction was weakly positive.

Autopsy revealed an adeno-carcinoma of the stomach; the cerebral arteries were markedly sclerosed. Microscopic examination of the spinal cord showed a typical combined degeneration in the posterior and lateral column.

The original diagnosis was that of senile paraplegia, but was later changed to subacute combined degeneration. Although no pathological evidence is given, the author suggests that the muscular atrophy was the result of a peripheral neuritis. Perhaps the most interesting feature of this case is the association of a gastric carcinoma with the blood picture of a pernicious type of anæmia, and a combined sclerosis of the cord. Although this association, which has been reported on other occasions, raises etiological points of supreme importance, the author does not discuss this aspect of the question. M. C.

PROGNOSIS AND TREATMENT.

[205] **Intravenous treatment of some epileptics with calcium chloride and gluco-calcium.**—E. Klein and E. Forcione. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 372.

Entirely negative results are described by the authors who found that neither was the number or severity of the fits reduced nor was there any sustained increase in the calcium concentration of the blood by the use of either substance.

R. G. G.

[206] **Experience with 2,000 intramuscular injections of neoarsphenamine in neurosyphilis.**—A. Gordon. *Amer. Jour. of Syphilis*, 1927, xi, 525.

The author considers that intramuscular injections of 0·15 grm. of neoarsphenamine, dissolved in 1 c.c. of fresh sterile distilled water and repeated either daily or on alternate days, constitute the ideal method of administering arsenical drugs in neurosyphilis. The cure is progressive, neural reactions are not encountered, and unpleasant sequelæ such as hæmoclastic crises and venous thromboses are not possible. When small doses are injected into the muscles overlying the scapula with a fine needle the treatment is practically painless.

J. G. Greenfield.

Psychopathology.

PSYCHOLOGY.

[207] **The psychology of rowing.**—P. E. Vernon. *Brit. Jour. of Psychol.*, 1928, xviii, 317.

In the rowing of the individual, psychological factors such as his mental attitude are of equal or greater importance than his strength or muscular skill: his style and work are greatly influenced, often unconsciously, by autosuggestion. This applies less to thoroughly experienced oarsmen. The nature of suggestion, namely, indirect mental influence operating through affective dispositions, is demonstrated in individual rowing. In the rowing of the group 'hint' rather than 'prestige' suggestion is of primary importance, since the relationship between members of a crew is one of equality, not of dominance and submission. The eight may profitably be regarded as an organism owing to the reciprocal reinforcement of every attitude in one member by all the others through the medium of the run of the boat. For a boat to go well and win its races there must be developed both a close physiological and psychological interconnection between members of the crew: the former consists in harmonious synchronization of the movements of every member, the latter is a sentiment—group self-respect. Each depends upon and conditions the other. The level of group self-respect rises and falls chiefly with the crew's ideas of the probable success and failure of their boat. It is greatly affected by coaching. The group life of the boat club and the rowing personality are well adapted to the development of the necessary *rapport* between members of the crew. The boat club is a society of a very primitive type. Observations also support a physiological type of theory of affect and do not support the concept of a general undifferentiated nervous or mental energy.

C. S. R.

[208] **The development and function of clothing.**—KNIGHT DUNLAP. *Jour. of Gen. Psychol.*, 1928, i, 64.

THERE have been but four important theories as to the origins of clothing. These are:

1. The modesty theory—which holds that clothing was originally donned to conceal the genital organs, from a sense of shame, modesty, embarrassment, or some other form of sexual emotion.

2. The immodesty theory—popularized by Westermarck and Havelock Ellis, maintains that the purpose of clothing in the beginning was salacious, designed to attract attention to the sexual organs, concealment increasing interest.

3. The adornment theory—that clothing begins in the desire to attract attention, not necessarily of a direct sexual sort.

4. The utility or protection theory—supposes that clothing had its origin in attempts to protect the body from injurious or unpleasant features of the environment.

The modesty theory has probably had the greatest vogue in modern civilization in spite of the various lines of evidence against it. Its falsity should have been evident long ago from the fact that oriental women have had to conceal their faces to be modest, while occidental women have not been under similar necessity; and the fact that native savages are often more modest than clothed Europeans. The immodesty theory is just as improbable, and the study of ancient and savage social conditions and clothing habits produces evidence against both the above theories. Clothing itself has neither modesty nor immodesty. It is merely the breaking of the established convention which makes it immodest. As regards the third theory, we may grant the human tendency to seek the limelight and the influence of this in the developing of clothing and adornment, but if the simple wish for conspicuousness were operating, the individual who should omit the ornament would be the most successful. Granted a clothing system, ornament may be expected to enter into it, but antecedently there must be another factor. The fourth theory has been in disfavour because it has not been easy to see how the most primitive clothing really affords any protection. The possible protection from climatic influences, or from thorns and brambles, is but a minor detail, and the author shows how the most characteristic types of costumes of savage races which flap with the movements of the wearer have the important function of protection from insect pests. Many of the other curious savage adornments are distinguishing social badges and the development of attire from that stage is largely a matter of habit and tradition. The association of clothing and modesty developed slowly and from these early stages the evolution of male and female clothing is different in various parts of the world. Apart from habit, another reason why we conceal our figure in clothes is to be found in sexual selection and competition. The writer in interesting detail points out that in looking back over the history of clothing and adornment we find a great deal which has been

effective in equalizing sexual competition, reducing the competition to a less deadly level. In some respects, at least, savages have been wiser than civilized peoples. Both physical and mental hygiene demands clothing that shall not transgress the limits of protective needs.

C. S. R.

NEUROSES AND PSYCHONEUROSES.

[209] **Studies in stuttering. II—Photographic studies of the voice in stuttering.**—L. E. Travis. *Arch. of Neurol. and Psychiat.*, 1927, xvii, 998.

In the method employed by the author Dorsay phonelescopes are used as optical levers for recording both the sound waves of the voice and the 100 double-vibration tuning-fork. The records are made on standard size motion-picture film and show a base line, a time line, and a voice line.

In unemotional, propositional speech records of the stutterer's voice show: (1) the presence of a vibrato and tremolo: (2) periods of tonal fixation, and (3) marked prolongation of tones. Other phenomena found in stutters are: (1) periodic fluctuation of breath pressure: (2) short periods of vibrations before the voice waves proper begin: (3) bizarre waves in the voice curve: (4) groups of oscillations of high frequency but low amplitude, and (5) long series of oscillations which are initiated extremely suddenly by a wave of much greater amplitude than any of its followers.

R. M. S.

[210] **Neurotic behaviour and the teaching of conditioned reflexes.**—A. G. Ivanov-Smolensky. *Amer. Jour. of Psychiat.*, 1927, vii, 483.

Neurotic conduct is the expression of disturbed equilibrium between cortical stimulative and inhibitive processes: either cerebral stimulation or inhibition predominates. The "psychic trauma," generally supposed to be the cause of the development of neuroses, from a physiological point of view is a task difficult for the balancing of stimulative and inhibitive cortical processes (differentiation or integration). The cerebral hemispheres being stimulated to activity by the lower parts of the nervous system (feeding, defensive, sexual, etc., and other unconditioned centres) at the same time meet intensive hindrances in biosocial surroundings; thus in the cortex a conflict of the stimulative and the inhibitive processes takes place, which produces an abrupt disturbance of intracerebral balance and a widespread irradiation of stimulation or inhibition (stimulative or inhibitive neurosis). The "dislodged complex" corresponds to the origination in the cortex of an inhibition, the result of an unsuccessful bio-adaptation. The cause of a neurosis is incongruity between the complicated nature of the surrounding world and the ontogenetic want of adaptability of the given cerebral hemispheres. The treatment of a neurosis must tend not only to remove cortical "inhibition spots" (dislodged complexes) but also to train the reflexogenous and balancing function of the cortex.

C. S. R.

[211] **The relation of occupation to migraine.**—W. ALLEN. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 131.

IT is pointed out that there is a generally accepted idea that brain-workers and the more cultured members of the upper classes are more subject to migraine than others. The author has tabulated the occupations of 400 migrainous patients and 1,000 non-migrainous patients and finds nothing to support the view that occupation has an influence on the occurrence of migraine.

R. G. G.

[212] **Clinical contribution to drug addiction: the struggle for cure and the conscious reasons for relapse.**—L. KOBB. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 22.

REAL drug addicts are possessed of a pathological nervous constitution with its inferiorities, pathological strivings, etc., and relapse is due to the fact that often only by the use of opiates can relief from these distresses be obtained. Now-a-days the laws restricting the sale of drugs prevent milder cases from resorting to drugs after they are once "cured," but the more severe cases revert again and again and so relapses seem to be more common. Nearly all addicts make sincere efforts to be cured in the earlier stages, but later the effort is at best half-hearted and only undertaken for purposes of expediency. The hope for cure wanes as time passes, and the force of habit, numerous impelling memory associations, and increasing physical dependence on opiates are added to the original pathological nervous condition. At the same time physical dependence on opiates is unimportant, as a cause for relapse during the first two or three years of addiction, in those who have been off the drug for two weeks or more. In some very nervous persons, with addiction of long standing, withdrawal of the drug may produce hysterical symptoms or hypomania which may last for several months.

R. G. G.

PSYCHOSES.

[213] **The significance of research into heredity for the diagnosis and prognosis of the endogenic psychoses** (Bedeutung der Erblichkeitsforschung für die Diagnose und Prognose endogener Psychosen).—HERMANN HOFFMANN. *Münch. med. Woch.*, 1926, 1104.

UNTIL recently work on inheritance of mental diseases was largely focussed on whether it was of the similar or dissimilar type: it has now been definitely proved that similar forms preponderate in heredity, but are not exclusively present. Thus the psychotic parents of cases of circular insanity show 69 per cent. of the same type, with 3 per cent. of the schizophrenic type, whereas among the psychotic parents of schizophrenics 52 per cent. were also schizophrenics and 21 per cent. manic-depressives. Thus schizophrenics appear

relatively less frequently in the heredity of manic-depressives than manic-depressives in that of schizophrenics. We should be very cautious in diagnosing manic-depressive insanity in the child of a schizophrenic parent, and in the author's experience when this does occur the other side of the family shows some cases of the manic-depressive type. On the other hand, there is quite a considerable probability that insanity in the child of a manic-depressive case may take the form of schizophrenia, though manic-depressive psychosis is the more probable form. Moreover, where such children do show the schizophrenic form, we find that either the other parent or his family shows schizoid or schizophrenic manifestations, or else that the manic-depressive psychosis is of an atypical form, either an agitated melancholia with anxiety and catatonic traits, or irritable querulous and paranoid ones, or else phases of true mania and depression superimposed on a foundation of schizophrenic traits. While true manic-depressive patients do occasionally have schizophrenic children, this is infrequent. Among the atypical circular psychoses we would include for our purpose all psychoses combining depressive and schizophrenic features, and the offspring of these are mainly definite and progressive schizophrenics. The same holds good for the paraphrenics, but less completely so for the paranoias, querulant and other, and senile paranoid psychoses. Circular psychosis is rare in the offspring of these, but similar paranoid psychoses occur as well as schizophrenia.

Leaving now the cases of direct inheritance, we find that a number of the endogenic psychoses show no morbid heredity. Others show it indirectly only, and prominent among these is the collateral inheritance in schizophrenia. For instance, take a patient suffering from a prolonged depression with a sensitive paranoid tendency without being definitely paraphrenic. There is no manic-depressive psychosis in the family, but a maternal uncle had a similar illness which developed into a definite schizophrenic terminal state after a few years. Collateral schizophrenic cases make the prognosis unfavourable especially when the mode of onset has been similar: whereas if, in the above case, the uncle had only had a real manic-depressive attack the prognosis in the doubtful paranoid depression would have been better, though more uncertain that the definitely bad prognosis with schizophrenic collaterals. Our material is not yet sufficient to give any definite indications. Where the inheritance is mixed all sorts of mixed forms may occur, and heredity can give little guidance as to the probable outcome in any given case.

Where brothers and sisters are psychotic the forms are more frequently similar, but dissimilar forms occur with fair frequency as might be expected when the inheritance is mixed.

Psychopaths of a definitely schizoid type are more likely to have schizophrenic than manic-depressive children, while if both parents are of the cyclothymic psychopathic type the children are considerably less likely to be

schizophrenics. Where the parents are psychopaths of different types, the children are more likely to have a psychosis of mixed form, while paranoid and querulant psychopaths tend to have schizophrenic children.

In conclusion, we may say that where the schizophrenic parents have had a psychosis of a mild and slowly progressing type, the children are more likely to have one taking a more rapid course, and of earlier onset by an average of 13 years: the children are unlikely to show a remitting type when that of the parent has been of the steadily progressing type.

M. R. B.

[214] **A study of the endocrine organs in the psychoses.**—BERTRAM D. LEWIN. *Amer. Jour. of Psychiat.*, 1927, vii, 391.

THE findings in the various organs were as follows. In general paralysis, extra-tubular fibrosis and exudate in the testes dependent on local vascular alterations: patchy fibrosis and exudate in the ovaries: rarely a lymphoid exudate in the hypophysis: frequently a lymphoid exudate and fibrosis in the adrenals; nothing specific or unique in the thyroid. In the cerebral syphilis group, occasionally changes such as those found in the general paralysis group. In the senile group, an exaggeration of the normal degenerative process of old age in the testes; except for arteriosclerotic changes, no constant findings in the other organs. In the arteriosclerotic psychosis group, the presence of a patchy, extra-tubular fibrosis in the testes seemed dependent on the presence of local arteriosclerosis; no special features in the other organs; arteriosclerotic changes. In dementia præcox, the lesions described in the endocrine glands were inconstant, and those which were most common (e.g., extra-tubular fibrosis in the testes, patchy fibrosis in the ovaries) were probably due to intercurrent chronic disease. The other psychiatric groups contained too few examples to allow of any anatomical and psychiatric correlation. The author emphasizes that he does not mean to assert the absence of any possible relation between endocrine organs and psychosis in those groups, such as dementia præcox, where the findings were essentially negative. He does, however, feel that there was no evidence in his material of such relations and for the dementia præcox group in particular he believes that his material was quite comparable to that on which several untenable hypotheses have been based.

C. S. R.

[215] **The functional psychoses as an evolution of psychic impotency.**—J. H. CASSITY. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 105.

THE author quotes several cases to illustrate his thesis that unfortunate environmental factors, whether they be micro-organisms, ill-treatment by parents, sexual traumas, nursing excesses, or what not, often seriously interfere with normal psychosexual development; and it is then that the individual is obliged to change his psychic attitude towards life in order to reconcile his

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actual or imagined sexual inadequacies with sociological and biological requirements. Further, it is in this shift and transformation that he assumes a different perspective from the average individual and hence becomes a neurotic or psychotic. The author regards this as affording an explanation of many of the reactions of paranoia, melancholia and general paralysis, and thinks that there may often be actual sexual weakness of a somatic type. He does not wish to advance this theory as all-embracing but thinks that impotence has more to do with psychotic developments than is generally believed.

R. G. G.

[216] **Endocrine and biochemical studies in schizophrenia.**—K. M. Bowman. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 483.

The author sums up the study as follows. The findings are not consistent with the constant presence of any definite endocrine disorder and do not suggest that a simple glandular dysfunction of a constant type is an etiological factor in schizophrenia. Rather they suggest that many functional disorders, closely linked up with the endocrine system, are frequently found and that schizophrenia is not a specific endocrine disease but may arise on a number of different bases. The one constant finding appears to be that a metabolic disorder of varying degree is nearly always present, as manifested in functional gastro-intestinal disorders and a tendency towards low basal metabolism.

R. G. G.

[217] **On irritability in manic depressive insanity** (Ueber Reizbarkeit im manisch-depressiven Irresein)—Eugen Kahn. *Münch. med. Woch.* 1926, 1314.

By irritability we understand a special emotional state with a sense of unpleasant inner tension apt to be discharged by emotional outbursts. Such states are common in physiological and pathological states, pregnancy, menstruation, menopause, in organic diseases, manic-depressive insanity and in neurotics and psychopaths.

In manic-depressive insanity we may divide such states into two groups—that of a constitutional lasting state, and a phasis form, though transition forms also occur. Among a small unselected group of manic-depressives there were 39·5 per cent. men and 34·1 per cent. women who showed this symptom, while many more showed characteristics of a kindred nature. Age and sex do not appear important factors. Phasis irritability is most often shown in the transition from a depressed to a manic phase, while prolonged irritability is shown in some chronic melancholias. The irritable types of melancholia show all grades of transition to the paranoid, with delusional ideas associated. Cases of mania or melancholia may have phasic irritability which seems to represent an abortive phase of the opposite type. It seems probable that irritability is part of a special constitution associated with but not an essential part of the

manic-depressive and cycloid inherited constitution, and it is probable that investigation of the inheritance of psychopaths would show this as a special type. States of nervous irritability may be hypomanic and hypomelancholic phases not recognised as such. All possible combinations of phasic and chronic and progressive irritability occur with all phases of manic-depressive and cycloid tendencies, and little is clear beyond their tendency to occur in combination. In some cases where manic and depressed states occur simultaneously the irritability seems to result from the conflict between stimulation and inhibition of volition. It is possible that this conflict causes the unpleasant tension which shows itself in irritability, the predominance of one or other resulting in manic or depressed phase, while lasting irritability results from a balance being maintained between them. Possibly endocrine and chemical conditions play a part; and alcohol or other drugs are often resorted to to allay the irritability. The author does not, however, regard irritability as part of the manic-depressive disposition, and thinks that it may play a part in many types of psychopathy and psychosis, so that each case must be treated as an individual and treatment be directed to adjustment of the whole personality and environment, so as to relieve the painful tension.

M. R. B.

[218] **The syndrome of mental automatism and its role in the formation of the chronic systematized psychoses: a review.**—P. BAILEY. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 345.

MENTAL automatism may be sensory, motor or ideo-verbal, and is regarded by de Clerambault (whose work is under review) as the basal factor on which a hallucinatory psychosis is built. Systematized delirium is the reaction of the subject's personality to the phenomena which take place within him and which he regards as extraneous in origin because they are foreign to all his usual system of experience. Sometimes the phenomena are not commented upon by the patient, in which case a condition of chronic hallucinosis results, but usually the patient reacts with a systematized delirium the nature of which depends on the strangeness of the phenomena, the intellectual make-up of the patient, his character and affective tone. The actual automatism is supposed to be due to a physicochemical alteration of cortical neurones, due to infection, intoxication degeneration, traumatism, etc.

R. G. G.

[219] **Heredo-congenital onychogryphosis, total alopecia, and schizophrenia.**—MAX SCHMIDT. *Acta Psychiat. et Neurol.*, 1927, ii, 122.

CASES of onychogryphosis of familial incidence are extremely rare; a familial anomaly comprising generalised alopecia and onychogryphosis is still rarer, only two instances being on record. Schmidt's paper concerns a remarkable family characterized by onychogryphosis, hypotrichosis, and schizophrenia. The nail changes were the most outstanding feature and were traced by the

author through five generations. Out of 115 members of this family 33 individuals had gross nail changes, and another nine showed a minor type of lesion. Males and females were both affected. Two sisters in this family together with their mother showed in addition a marked onychogryphosis, a generalized hypotrichosis and schizophrenic symptoms. In this case not only had the mother suffered from a depressive psychosis, but her husband also (who did not belong to the family) was of psychopathic temperament and stock.

The author refers to an interesting community living in the south-west provinces of France known as the Cagots. Of obscure origin, the Cagots have always been social outcasts: their importance in this present connection lies in their physical conformity: cretin-like in appearance, they are of a yellowish-pale complexion; the hair of the head and body is scanty and the nails are like talons. The Cagots have been thought to be descendants of lepers; others have believed them the surviving members of the Visigoths, and trace the word "cagot" from "canis gothicus."

M. C.

PSYCHOPATHOLOGY.

[220] **The unmarried mother; a socio-psychiatric viewpoint.**—HENRY C. SCHUMACHER. *Mental Hygiene*, 1927, xl, 775.

IN approaching the subject of illegitimacy, five factors at once clearly present themselves. In the first place, these unmarried mothers are mostly young; over 75 per cent. are under twenty-one years of age. Secondly, most of these mothers come from the economically inferior strata of the population, the majority of those employed being domestic servants or semi-skilled factory workers. In the third place, to a large extent they are of inferior mentality. On this point, as well might be expected, there is the greatest divergence in the available statistics. In the fourth place, over half of them have previously been delinquent, a third of them previously immoral. Lastly, well over half come from homes in which there are immorality and alcoholism, poverty and dependency, absence of parental training.

Evidence shows plainly that there is no justification in assuming that of necessity a causal relationship exists between sex delinquency and mental pathology. There may be an unduly and disproportionately strong sex urge from internal glandular imbalance: there may be lack of normal inhibition; or mental defect. There are individuals who are quite normal physiologically and intellectually, but who engage in illicit sex relationships as an expression of a definite behaviour tendency. Frequently such an individual uses her sex life to overcome or to compensate for thwartings of desires or of activities in other directions, or to gain consideration and through it expression of other desires and interests. One must not overlook that type of girl who deliberately and consciously chooses to gratify her passions. Here the behaviour is not due to unconscious, repressed complexes. Such a girl's conduct depends solely

upon her acceptance of a low ideal. Her actions are left to the mercy of her impulses and illegitimate motherhood is but accidental to illicit sex relations Lastly come the psychotic and the more serious psychoneurotic disturbances. Here the individual definitely fails in her attempt at adjustment along socially acceptable lines. Often sex promiscuity in such individuals is but an expression of the breakdown of the normal inhibitions or a freeing of former repressions due to the mental ailment. C. S. R.

[221] **Psychic factors in juvenile delinquency.**—FREDERICK H. ALLEN. *Mental Hygiene*, 1927, xl, 764.

HERE is discussed the individual's struggle to attain self-respect, self-confidence, and an adequate sense of his own individuality. The failure to attain this goal by means of socially constructive types of activity may lead to any one of the various manifestations of abnormality, among them being various types of delinquency. Failure to attain self-respect and self-confidence usually leads to the development of unhealthy compensatory activity which, if not corrected, may form the basis for a criminal career or for one of the varied nervous and mental disorders. Delinquency is one of the commonest forms of compensatory effort made by individuals to overcome some of their handicaps. In analyzing the histories of sixty stealing cases studied in a child-guidance clinic, it was found that in nearly half of them the stealing and associated activity were related definitely to feelings of inadequacy which had their roots in various factors in the life situation of the child. Common among these factors were physical characteristics which stamped the individual as being different, such as obesity, speech defects, undersize; mental defects of varying degrees; certain habits, such as enuresis, masturbation; racial prejudices; presence of more attractive and gifted brothers and sisters; immorality and desertion of parents; economic factors; failure to achieve a healthy emancipation from parents; and repressive discipline. Illustrative cases are given and it is pointed out that the treatment must be directed toward one or more of three things: (1) the elimination of one or more of the sources that are feeding a sense of inadequacy. As many of these factors are fixed, it is necessary to resort to another approach; (2) changing the attitude of the individual and others in his environment toward the source of the difficulty—a procedure that usually involves the third step, namely: (3) redirecting the activity into more socially constructive channels, strengthening the constructive assets of the individual, broadening interests, and creating opportunities for new ones. A fourth and somewhat negative line of approach is the avoidance, as far as possible, of creating new sources of difficulty by mistaken methods of handling those already developed. Something besides mere punishment and correction is necessary. The measures that are necessary in each case can be determined only after an understanding of the dynamic factors has been reached and a plan evolved that will strike at the roots of the problem. C. S. R.

[222] **An investigation in illustration of the importance of neurology to mental deficiency.**—Erik J. Larser. *Acta Psychiat. et Neurol.*, 1927, ii, 71.

Critical of previous work on the heredity of mental disorders which fails to distinguish clearly between primary and secondary mental defect, the author undertook an enquiry into the importance of hereditary factors in the primary amentias. Out of a total of 100 juvenile asylum patients, evidence of organic nervous disease was found in 70, as opposed to 29·7 per cent. of cases of children from special schools Grade 2, and 20·9 per cent. of cases from special schools Grade 1.

The author is inclined to place importance upon the role of birth injury in the production of mental disorders and cites the presence of facial palsy as evidence of such trauma. Although admitting that signs of bilateral disease are almost always demonstrable in apparently unilateral cases, he still suspects birth injury, for he says, "it is not to be expected that a birth lesion should be unilateral."

M. C.

[223] **Blood groups in mental diseases.**—F. Pròescher and A. S. Arkush. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 569.

The authors as a result of their investigations envisage a time long ago when no separation into races had occurred and all individuals were universal donors. The foetus passes through this stage to-day just as in other respects it repeats, in rough form, its phylogenetic development. With this picture of the primitive universal donor in mind it would seem that the crudest and least highly developed of nervous systems might belong in general to Group I members. This simple system with its more undifferentiated parts might be expected to be less susceptible to injury by those higher factors, loss of which stigmatizes the person concerned as insane. In other words Group I members should be least susceptible to insanity, and this is actually the case. The authors suggest that these findings, added to such facts as inherited insanity, are strong indications for an organic basis of insanity and that is it a matter of race and structure and not of infection caprice or environment (though the power of environment to demonstrate or precipitate insanity is well known).

R. G. G.

[224] **The adrenalin glycaemic curve as a diagnostic aid in psychiatry.**—Harold Gordon, Jessie M. Ostrander, and Stella Counsell. *Amer. Jour. of Psychiat.*, 1927, vii, 183.

These investigators found definite group curves in manic-depressive and in dementia præcox cases free of somatic disease. In the manic-depressive psychoses, the curves showed a relatively well-defined peak and delayed return of the blood sugar to the primary level. In dementia præcox cases the curves showed more or less of a plateau, a less sharply defined peak and a less marked delay in returning to the fasting level. In a small series of cases with hepatic

disease they also found a distinctive blood sugar curve. In these cases the curves tended to become flat and showed a terminal rise. Similarly, in the endocrinopathies flat, rising curves were obtained. This test showed promise both as a means of confirming psychiatric diagnosis as between manic-depressive and schizophrenic cases, and as a method of determing the glycogenic function of the liver.

C. S. R.

PROGNOSIS AND TREATMENT.

[225] **The treatment of neurosyphilis and paresis with malaria.**—H. GOLDSMITH and R. ROCKWOOD. *Amer. Jour. of Syphilis*, 1927, xi, 165.

THE authors have treated 14 cases of paresis and cerebrospinal syphilis with double tertian malaria, followed by intensive arsphenamine treatment. Eight of the patients have returned to their usual work. Two died, one from syphilitic heart disease during an attack of ague and the other from cystitis and terminal pneumonia. They recommend the treatment for selected early cases of paresis.

J. G. GREENFIELD.

[226] **The malaria treatment of general paresis.**—A. FERRARO and T. C. C. FONG. *Jour. Nerv. Ment. Dis.*, 1927, lxv, 225.

THE results claimed in the published accounts are tabulated, and the methods employed by the authors and others are fully discussed. The effect on individual symptoms and on serological reactions is then dealt with, as is the relationship of results to the special technique employed.

Finally the possible explanations of results are reviewed and the authors suggest that the action of the raising of the body temperature in destroying the spirochaetes is probably responsible for improvements.

R. G. G.

[227] **The intraventricular treatment of paresis.**—R. CESTAN and M. PERES. *Jour. Nerv. Ment. Dis.*, 1927, lxvi, 227.

A FAIRLY extensive experience of injecting salvarsan intraventricularly leads to the conclusion that when the ventricular fluid is rich in albumin and cells the physician can with impunity inject up to 6 mgm. of salvarsan. In proportion as the ventricular fluid is or becomes less rich in albumin and cells the dose must be rapidly diminished to 1 mgm., which is always tolerated. Although serological reactions are often considerably modified by this treatment neither the symptoms nor the clinical course of the disease seem to be much altered.

R. G. G.

[228] **Afenil treatment in schizophrenia** (Zur Afeniltherapie der Schizophrenie).—U. BERNDT and K. KULLE. *Münch. med. Woch.*, 1926, 1191.

THIS is an account of a trial of afenil, which was given as advised by Dodel, 10 c.cm. intravenously every three days, 20 to 30 doses being given in each case. Thirteen cases were tried, varying in age between 18 and 50 and of all types, early, relapsing and chronic; all were definite schizophrenics.

Of the 13, two only showed any benefit which could be attributed to the treatment; of these one, a case of gradual development with attacks of hallucination and excitement, improved greatly, gained insight, and was discharged after remaining quiet and accessible for two months. The other was that of a patient who had had a previous remission of four years' duration, and who relapsed after an improved period of nine months following the treatment. The rest showed no benefit at all, though many of them were early first-attack cases, while others had had previous remission. The authors conclude that afenil is shown by their results to be harmless, but quite ineffective even as an aid to improvement in schizophrenia.

M. R. B.

[229] **The need for co-operation between the legal profession and the psychiatrist in dealing with the crime problem.**—WILLIAM A. WHITE. *Amer. Jour. of Psychiat.*, 1927, vii, 493.

SOCIETY has the right to segregate the dangerous antisocial types so long as they continue dangerous. This means largely doing away with fixed sentences, at least for certain types of crime, and making the return to freedom conditional upon some change in the individual that gives one a right to suppose that perhaps he will function more effectively as a social unit than he has in the past. It means the elimination of punishment as a vengeance motive and its retention only if used for definitely constructive ends for conditioning conduct; also the gradual transformation of prisons into laboratories for the study of human behaviour and the conditioning of human conduct. The concept of responsibility, which is largely a point of metaphysics, should be discarded. It would be an excellent thing for those legal authorities who have to deal with crime to serve internships in psychiatric clinics and prisons in the course of their educational preparation, just as physicians do in their various medical specialities.

C. S. R.

Reviews and Notices of Books.

Conditioned Reflexes. By I. P. Pavlov. Translated by G. V. Anrep. London: Oxford University Press: Humphrey Milford. 1927. Pp. 430. Price 28s. net.

Pavlov's experiments are undertaken entirely from the physiological aspect and the unit on which his researches are based is naturally the reflex. He advances arguments to show that the so-called instincts do not differ in their nature from the more complex reflexes and that all mental phenomena may be described in terms of reflex response. The reflexes involving the cerebral cortex are characterized by what is called signalization and the intervention of distance-receptors. They are acquired after birth and constitute the conditioned reflexes as opposed to the unconditioned inborn reflexes. The study of the circumstances governing these conditioned reflexes led to the study of inhibition. This showed that the cortex was concerned not only with positive conditioned reflexes but with negative or inhibitory conditioned reflexes, and that a sensory stimulus may result in inhibition as well as excitation. Various types of inhibition are described and this leads to a discussion of experiments illustrating the analyzing and synthetizing activity of the cerebral hemispheres. Irradiation and concentration of nervous processes are the subject of further investigation and this is followed by evidence for the identification of sleep with internal inhibition and all the implications associated with this. The effect of damage to the cerebral cortex on the excitations and inhibitions of reflexes is discussed as length and the difficulties and unsolved problems associated with conditioned reflexes are dealt with.

The last lecture is devoted to applications to man. While these applications must be made with caution Pavlov believes that the "neurasthenic" type of neurotic corresponds to the excitable type of dog, characterised by prolonged periods of exhaustion, since the nervous system is always subjected to continuous wear and tear. The "hysteric" type of neurotic is said to correspond to the inhibitable type of dog with brief periods of excitability when control is in abeyance. Many of the experiments seem to suggest possible methods of therapy by developing internal inhibitions. The special form of inhibition involved in hypnosis is referred to and the analogy of suggestion with many of the conditioned reflexes obtained in the experiments is remarked.

The English-speaking medical public owe a deep debt of gratitude to the Royal Society for making possible, and to Dr. Anrep for so admirably carrying out, the translation of this work, which is without doubt the most important contribution to the physiology of the nervous system since the publication of Sherrington's classic in 1906.

R. G. G.

Mental Disorders: A Handbook for Students and Practitioners. By Hubert J. Norman, M.B., D.P.H., Medical Superintendent, Camberwell House; Lecturer on Mental Disorders, Westminster Hospital Medical School. Edinburgh: E. & S. Livingstone. 1928. Pp. 463. Price 14s. net.

Any textbook destined for the hands of students and practitioners and dealing with the problems of the mind diseased must have one virtue at least which may cover a number of sins of compilation, and that is—readableness. A dull book on mental disorder would seem a paradox, yet such are known, unfortunately. Those who are familiar with Dr. Norman's published work will look in this handy little volume for readableness and literary allusion and pleasantly humorous comment, and they will not be disappointed. To our mind these traits add materially to its interest; the student most of all should be indebted to its author, for here he has psychiatry without boredom.

Dr. Norman omits prolegomena dealing with normal psychology and presents the reader at once with his conception of the way to approach psychiatric questions. He leaves him in no doubt as to what this is; mind is a term for the functioning of the brain, just as respiration is a term for the functioning of the lungs, although what has become practically axiomatic in neurology "is being but slowly accepted in the pseudo-science of psychology." The classification of mental affections herein adopted is based largely on clinical criteria and if in a sense old is none the less of proved worth. Underlying the psychoses is constitutional instability; the psychoneuroses are separable therefrom: then come symptomatic disorders, that is, disorders arising on known toxic, structural or other alteration of the nervous system. Only after this sufficiently adequate description of clinical varieties is the reader given a discussion of the normal functioning of the mind and a brief sketch of the physiology of the cerebral cortex. Pathological, therapeutic, and legal considerations are in turn developed.

Apart from its more purely technical side this compact handbook contains no little information of a historical and literary kind which indicates the wide range of its author's reading and which will delight all those who like to follow bypaths of medical lore.

Epilepsy: Comparative Pathogenesis, Symptoms, Treatment. By L. J. J. Muskens, M.D., Amsterdam. Foreword by Sir Charles S. Sherrington, O.M. London: Baillière, Tindall and Cox. 1928. Pp. 435. Price 27s. 6d. net.

We have recently reviewed in this Journal the original German edition of Dr. Muskens' carefully written work on epilepsy, and have alluded to its basis in personal experimental researches on the production of epileptic phenomena in animals and to the general theory of the author, that myoclonic reflexes constitute as it were the framework of the epileptic attack. In its English edition the book will be found to supply what is rather a want in

medical literature at present, for it furnishes a modern setting for the age-old problems of epilepsy and is replete with information of the most varied kind on all of the many aspects of the affection. Some 55 diagrams and illustrations add to its value.

Selected Papers of Karl Abraham. With an introductory memoir by ERNEST JONES. Translated by DOUGLAS BRYAN and ALIX STRACHEY. The International Psycho-Analytical Library, No. 13. London: The Hogarth Press. 1927. Pp. 527. Price 30s.

THIS large volume of twenty-six separate psychoanalytical papers and essays, mainly arranged in chronological order, contains the whole of Abraham's more important work dating from 1907 to the year of his death (1925). In the introductory memoir the salient features of these contributions are well reviewed and appraised. It is apparent at once from a glance at the subjects herein dealt with that they cover a wide field and it is equally evident when they are read that they are illuminating in character. Because of their extensive variety Ernest Jones groups them under the headings of childhood-sexuality, clinical subjects, general topics, and applied psychoanalysis. Clinically, special interest centres round the work done on the psychopathology of dementia præcox and manic-depressive insanity. Much stimulating material is also found in the analytic work dealing with hysterical dream states, locomotor anxiety, war shock cases, and alcoholism. Those who are in the best position to judge would probably select Abraham's findings on oral erotism as his most valuable contribution. Those who are sufficiently well versed in the subject to profit from reading this book and are interested in psychoanalytic conceptions will find much here of great value. A complete bibliography of all Abraham's scientific publications is appended.

Problems in Psychopathology. By T. W. MITCHELL. International Library of Psychology. London: Kegan Paul, Trench, Trubner & Co., Ltd. 1927. Pp. 190. Price 9s. net.

DURING late years the ground more or less covered by the author has been dealt with by others but such a difficult subject could not possibly be more ably presented than Dr. Mitchell has done in these pages. There are few authorities who are capable of dealing with the conflicting views of psychopathology so concisely or with such clarity, and of summing up in a broad and critical scientific spirit the values of the various schools of thought. Pre-Freudian psychology is first briefly dealt with, but as it is considered that the psychoanalytic approach is most worthy of consideration the main part of the volume is devoted to an exposition of Freudian theories. The chapters on the theory of the libido, the instincts, the ego, and the neuroses, are models of lucidity, and herein Freud's more recent work is discussed. After dealing with the applications of medical psychology, there is a survey of its doctrines and the varying concepts of the "post-Freudians," Jung and Adler, are shortly

and clearly discussed. Dr. Mitchell states that he has taken psychoanalysis as his main theme because " it is the dominating influence in all the work on medical psychology that is being done in this country at the present time. It is the centre around which all other schools of mental pathology and mental therapeutics revolve. Those who are hostile to many of its tenets cannot get away from it, although they cannot accept it. It thus remains a force which has important effects far beyond the range of influence exerted by the relatively small group of workers who unreservedly acknowledge themselves adherents." The book gives one an excellent insight into the problems of psychopathology and for this purpose can be highly recommended.

Principles of Abnormal Psychology. By E. S. Conklin, Professor of Psychology in the University of Oregon. London: George Allen and Unwin, Ltd. 1928. Pp. 457. Price 15s. net.

Professor Conklin's book is intended for students of psychology, though it deals almost entirely with psychopathology in the widest sense of the term. This extension of the realm of what is ordinarily understood by psychology is characteristic of the day; in other years the student of psychology took a course which was a constituent of the curriculum for an arts degree; now, the tendency obviously is for his instruction to be coloured with a most distinctly medical tinge. If he is to be familiarised with the psychoses, psychoneuroses, shell-shock, abnormalities of sleep, and what not, we may ask what becomes of the science and art of psychiatry, and whether psychopathology should come under the province of the physician, or of the psychologist who may be ignorant of the physiology of the brain in health and disease. The question is important and likely to become more so, and on its solution much of good or ill for the future of medicine is calculated to depend.

The volume under review is replete with medical cases (apt and informative enough), yet we are told by the writer that psychology and medical practice are two distinct and separate things which are too often confused. This statement is open to considerable criticism if we believe that the brain is the organ of mind. In our opinion discussions of the mental affects of drugs, of feeblemindedness and idiocy, of disorders of sleep, of hysteria, paranoia and epilepsy—such as are well set forth in the author's pages—belong to medicine, to psychological medicine if that is thought a better expression, and to deal with them properly medical training is a *sine qua non*. As physiology is part of the medical student's course, so ought psychology to be; not otherwise can he become a soundly trained psychopathologist. Professor Conklin deals further with phenomena not usually regarded as having a medical bearing, viz., those connected with spiritism, telepathy, genius, and so forth, and his discussion is couched throughout in a lucid and readable style. Many of his comments on current theories are peculiarly shrewd; what may be termed the viewpoint of common sense receives full consideration. Numerous references to the literature are given at the close of each section.

Studies in Psychology. By WILLIAM ELDER, M.D., F.R.C.P.E., Consulting Physician to Leith Hospital. London: William Heinemann (Medical Books) Ltd. 1928. Pp. 212. Price 8s. 6d. net.

THESE interestingly written essays deal with such mental phenomena as memory, emotion, consciousness, sleep, dreams, symbolism, somnambulism, and are both sound in theory and facile in expression. The author believes patently in the approach to psychology from the side of biology and physiology and is convinced of the futility of psychological hypotheses if they can be shown to run contrary to observed facts of these latter sciences. This, of course, is in a sense to compare incomparables; nevertheless, his contention has its value, for it is in accordance with recent trends in thought and research which are serving to bridge the assumed gulf between the neural and psychical spheres.

The analysis of the phenomena of the above-mentioned groups is conducted in a reasonable if comparatively simple fashion; controversial points are not tabooed yet are not accorded any profound examination. Hypotheses associated with the names of Freud and others of the school of psychoanalysis are discussed fairly if superficially, and on the whole unfavourably. The chapter on sleep is possibly the least adequate in the book; discarded views are given some attention while the work of Pavlov is referred to briefly but not in respect of his experiments on sleep, his collected volume on conditioned reflexes making its appearance when the author's book was already in the press. Unsatisfactory, too, are some of the arguments in regard to the seat of the emotions; the assumption that "the collection of the neurones into ganglionic groups in the optic thalamus may be due to a primary grouping of the various emotions" is contrary to the facts of both neuropathology and experimental physiology. Nor can we understand the view that "the extrapyramidal tract (*sic*) consists of fibres passing down from the corpus striatum to the sympathetic ganglia" (*sic*). This remarkable statement will not bear investigation. There is a certain weakness in the author's knowledge of present-day neurological problems which rather detracts from the force with which he maintains his general thesis, even if with it we are more or less in sympathy.

The Constitutional Factor in Disease. By ARTHUR F. HURST, M.D., F.R.C.P., Physician to Guy's Hospital. Psyche Miniatures: Medical Series, No. 9. London: Kegan Paul, Trench, Trubner & Co. 1927. Pp. 93. Price 2s. 6d. net.

FOR the neurologist the chief points of interest in this little volume are concerned with subacute combined degeneration of the cord, and with epilepsy, asthma, and migraine. In respect of the former Dr. Hurst urges its constant association with achlorhydria and malignant anæmia, and stresses its frequent occurrence in families one or more of whose members reveal signs of achylia gastrica—an "inborn error of secretion." Obviously, this view can be proved or disproved only by patient accumulation of clinical data; we do not think

that up to the present it has received universal support however impressive in some instances of the spinal affection the association appears to be. The other conditions alluded to above are classed by the writer as paroxysmal neuroses, whose basis is the existence of some constitutional and often inherited excitability—an "irritable centre"—in one or other part of the nervous system. It is doubtful if this view is wholly deserving of credence, particularly because of the ready appearance of epileptic phenomena in normal individuals (as in normal experimental animals) should circulatory, toxic, or other factors come into being.

Medicine: and the Man. By MILLAIS CULPIN, M.D., F.R.C.S. Psyche Miniatures: Medical Series, No. 7. London: Kegan Paul, Trench, Trubner & Co. 1927. Pp. 67. Price 2s. 6d. net.

DR. MILLAIS CULPIN tilts at current conceptions of "functional" nervous affections, and his strictures contain a considerable element of truth, far though they are from being novel or new. Seeking in his turn a more satisfactory definition of what is understood by a neurosis, he takes it to indicate "a disturbance of the intrinsic function of nerve tissue," and appeals for its avoidance if we have to deal with the *behaviour* of the person or patient concerned. It should be restricted to disorders that can be described in terms of physiology. Thus understood, it appears to us to come near to standing for any and all nervous symptoms, which clearly are the exteriorisation of neural dysfunction, and so dangerously close to an unspecific and meaningless term. Dr. Culpin possibly foresees this objection, for he declares that a neurosis is diagnosed "by the recognition of associated symptoms of mental disturbance, that is, by the coexistence of a minor psychosis." Yet by way of illustration he quotes a case of telegraphist's cramp which "was not truly a neurosis: it was a minor psychosis, a disturbance of behaviour like a stammer." But as the patient had palpitation and sweating also, these "can rightly be called neuroses, since they are disturbances of intrinsic nervous function."

We do not see that Dr. Culpin's exposition makes for either clinical or physiological clarity: but we are at one with him in his complaint about the vagueness of present-day teaching where "functional" affections are concerned. Through this essay runs a thread of somewhat heavy and sardonic comment which we consider rather regrettable: all of us make mistakes in diagnosis occasionally, and if, as Dr. Culpin says, "more than once or twice the surgeon was called on to open an 'acute abdomen' the owner of which returned to duty within a few hours," he might remember with advantage that myasthenics and patients with cerebral tumour have been submitted to useless psychotherapy, also more than once or twice. Extracts of organs, "mentionable and unmentionable, that form a valuable by-product of Chicago meat-works," have their place, even if on occasion the physician prescribing them sees in their use a way of escape from an unsolved problem.

Clinical Neurology for Practitioners of Medicine and Medical Students (largely based on the book by Prof. Dr. HANS CURSCHMANN). By EDWARD A. STRECKER, A.M., M.D., and MILTON K. MYERS, B.S., LL.B., M.D. London: H. K. Lewis & Co. 1927. Pp. 410. Price 15s. net.

THIS book is based upon a similar work by Professor Hans Curschmann, with the addition of new material by the two American authors. Intended as a "brief clinical exposition of neurology," it may claim to some success in this respect, though its value is marred by the dogmatic and sweeping character of certain pronouncements. Such bald statements as "the globus pallidus and its projections preside over automatic and associated movements," or, "epilepsy is a dreadful disease," may be selected as examples of a misplaced dogmatism. Many minor points catch the critical eye. Thus to dismiss the treatment of trigeminal neuralgia by alcoholic injections in the sentence, "Alcohol has been injected into the Gasserian ganglion" seems inadequate, even in a small textbook. Graves' disease and myxœdema are as a rule so well treated in text books of internal medicine that their somewhat sketchy description here seems hardly justified. The reader may justly be irritated by coming upon such vague phrases as, "In myasthenia we must deal with the possibility that the endocrine disturbances are only the co-ordinated syndrome in a general dysplastic state of affairs." We cannot approve the treatment which has been recommended for Raynaud's disease of "making fine incisions into the affected part and sucking up the cyanotic blood in a water pump," nor can we find any justification for the suggestion that resection of the sympathetic would be of value in the treatment of facial hemiatrophy.

A. F.

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